1981

A Comparative Radiographic and Histologic Analysis of Experimentally Produced Pulpo-Periapical Lesions in the Dog

Lance W. Crawford
Loyola University Chicago

A COMPARATIVE RADIOGRAPHIC AND HISTOLOGIC ANALYSIS OF EXPERIMENTALLY PRODUCED PULPO-PERIAPICAL LESIONS IN THE DOG

By

Lance W. Crawford, D.D.S.

A Thesis Submitted to the Faculty of the Graduate School of Loyola University of Chicago in Partial Fulfillment of the Requirements for the Degree of Master of Science

June

1981

DEDICATION

To Vicki, my wife and best friend,

and Russell and Jonathan, our children,

for their sacrifices which have made possible

the realization of a dream.

ACKNOWLEDGMENTS

To Dr. Franklin S. Weine, my friend and mentor, I thank you for sharing your knowledge, experience, and most of all patience with me.

To Dr. Norman K. Wood, I thank you for teaching me how to critically analyze scientific information during the course of this research project.

To Dr. Thomas E. Emmering, I extend my gratitude for your continued guidance throughout this study.

To Dr. Marshall H. Smulson, as Chairman, Department of Endodontics, for allowing me to pursue higher education.

To Dr. Terry L. Kippa, Dr. Alan T. Azar, and Dr. Richard A. Kohn, my colleagues, I express my appreciation for your friendship and help along every step of the way.

VITA

The author, Lance W. Crawford, was born in Joliet, Illinois on August 25, 1949.

He obtained his elementary education in the public school systems of Peoria, Illinois, and Evergreen Park, Illinois. In 1967, he graduated from Evergreen Park Community High School.

A Bachelor of Science degree was earned in 1971 from the University of Iowa before entering Loyola School of Dentistry in the same year. He received the degree of Doctor of Dental Surgery in 1975.

From July of 1975 until July of 1979 he pursued a career in the practice of general dentistry in rural Iowa. In August, 1979, he returned to Loyola University School of Dentistry and began a dual course of study leading to a Certificate of Specialty Training in Endodontics and the degree of Master of Science in Oral Biology.

TABLE OF CONTENTS

LIST OF TABLES

TABLE PAGE

LIST OF FIGURES

CHAPTER I

INTRODUCTION

A thorough understanding of the anatomy and histology of pulpo-periapical lesions is important in dentistry and specifically in endodontics. The dental radiograph, an important diagnostic tool, is unable to demonstrate existing conditions at the periapex when an incipient lesion is present. If the radiographic appearance of an incipient pulpo-periapical lesion were diagnostic of the actual disease process in every case, then the clinician's job would be considerably easier. In fact, however, the dental radiograph is of limited value in the detection of subtle bone density differences attributable to the demineralization associated with the formation of pathologic tissue.

The opinion that bony lesions actually exist before they appear radiographically is commonly acknowledged. A better understanding of the anatomy and histology of this area might help in our understanding of the disease process, its progression, and ultimately the treatment of same.

Therefore, it is the purpose of this study to analyze the radiographic and histologic appearance of induced pulpo-periapical lesions in an acceptable animal model. Any comparisons which can realistically be applied to the human counterpart may further our understanding of the pulpo-periapical lesion.

The availability, relative low cost, and reasonable size of the Beagle dog make it an appropriate candidate to test the applicability of this model to research of this nature.

CHAPTER II

REVIEW OF THE RELATED LITERATURE

A. HISTORICAL BACKGROUND OF RADIOLOGY AS RELATED TO DENTAL BONY LESIONS

In 1895, Roentgen discovered the x-ray. The following year William James Morton displayed some of the earliest dental radiographs. At approximately the same time, C. Edmund Kells gave the first clinic on dental radiology. In 1899, Dr. Kells may have first used radiography in endodontic therapy.[1] With the advent of dental radiography, previously unnoticed periapical pathoses became apparent to the medical profession.[1] This information did nothing to prevent the focal infection theory. However, as early as 1918, Coolidge and others, who had always related periapical infection to diseased pulps, were studying the limits of radiology on dentistry.[2] They observed that difficulty existed in determining when the periapical tissues were first involved coinciding with a diseased pulp. Even then the radiograph was the diagnostic aid depended upon to determine periapical involvement unless clinical evidence should manifest itself first. In the same article, Coolidge reported that chronic abcesses lent themselves to more periapical involvement and those radiolucencies are then more readily apparent. It is important to note that at this early date Coolidge realized that dead and/or infected pulps may have caused a problem before the radiograph could confirm the presence of the cause or effect. In spite of

these early advances, Coolidge condemned teeth whose pulps caused large areas of rarefaction.

In the same year, Nyman[3] stated that along with destruction of cancellous bone due to a necrotic pulp, a sclerosis of the cortical region will proceed. This sclerosis shielded the actual lesion from radiographic detection.

Miller and Peltzer[4] (1939) attempted to develop a guide for radiographic evaluation of bone loss in periodontal disease. They believed the rapidity of bone destruction was inversely proportional to the density of the alveolar bone. They also noted that the absence of lamina dura indicated the presence of osseous changes.

Several authors have questioned the value of radiography in the diagnosis of periodontal disease.[5,6,7,8] Barr[5] expressed the opinion that the only diagnostic information produced by x-ray films were conditions involving pathologic changes in the density of hard tissues. He also noted that the angle of the radiograph to the object may obscure the examiner's ability to see the pathosis. Very often he found the first evidence of pathosis to be buccal or lingual to the tooth in question. Its detection in those positions would be doubtful. Barr also indicated other problems in observing early pathosis on the radiograph. Dimensional accuracy of involved structures was often sacrificed due to limitations on the ability to place the film parallel to the object. He also described technical shortcomings of the film itself (image distortion, artifacts, etc.) as detractable items in diagnosis of pathosis. Furthermore, Barr found that density differences of as much as 0.31

could not be discerned in unmounted films while the difference between carious and non-carious dentin or enamel was only 0.20. Since properly mounted films allow detection of differences as little as 0.03, it was apparent that proper mounting was imperative for best results in diagnosis. This information was only of value on a comparative basis because no unit of measurement was designated in the paper.

Bohannan and Saxe[6] wrote that the diagnosis of periodontal disease could not be made from the radiograph alone. Far more important was the clinical examination with a periodontal probe. The radiograph, being a two-dimensional recording of a three-dimensional complex, had limitations. Specifically, the dental roentgenogram did not have the ability to record any usable soft to hard tissue relationships. Calcified deposits, even in large amounts, are not accurately portrayed on the radiograph due to the superimposition of structures. Also, they believed the amount of mobility of a tooth was a far more accurate measure of the severity of the lesion in occlusal traumatism than was the radiographic picture of the periodontal membrane space. Bradley[7] stated that a much overemphasized point was the early detection of periodontal disease by means of dental x-rays alone. He believed that since soft tissue changes were not discernable by x-ray examination, they could not solely be relied upon for clinical diagnosis. The author showed that pocket depth absolutely could not be determined by x-rays since the epithelial attachment was made up of soft tissue. In agreement with both Barr[5] and Bohannan and Saxe[6], Prichard[8] stated that regardless of roentgenographic findings, the patient must be examined clinically before a diagnosis of

periodontal disease could be made. Even then, only the parallel (right angle) technique was of any value in the diagnosis of periodontal disease by radiography.

Conversely, other writers[9,10,11,12] have stressed radiographic interpretation as essential to the adequate diagnosis of periodontal conditions. Ball[9] stated that, in the great majority of cases a more accurate diagnosis could be made from excellent radiographs than by all other methods of investigation combined, the emphasis being placed on the quality of dental radiographs. Frohlich[10] agreed by writing that roentgenograms, properly evaluated, were the best aid in diagnosing all periodontal diseases.[11] In referring to periapical disease, Selecky stated several findings including the claim that the radiograph served to "...discover, confirm, classify, or localize disease." He believed it to be of paramount importance to know the normal histology of the particular oral structure involved to recognize pathologic changes. His description of a pathologic condition indicated that salts must be unbalanced, or a part of the bone destroyed before it could be shown radiographically, since calcification and resorption of bone affect radiographic density. Also, he stated that marginal thickening of the periodontal membrane was a fore-runner of the penetrating condition and so the radiograph was an invaluable aid. However, it had to be correlated with clinical findings in order to properly diagnose pathosis. In 1958, Goldman and Cohen[12] found that the radiograph could be of great aid in the demonstration of the presence of buccal and lingual as well as proximal walls in a pocket occurring in the interdental area.[1] So, in 1918

and even into recent years[3] it has been realized that the radiograph is but one diagnostic procedure among several which, when collectively employed allowed the most reliable diagnosis of pathologic change.

B. THE STUDY OF BONE AND LESIONS OF BONE

In 1941, Ardran[13] attempted to determine quantitatively the amount of bone destruction possible without its being detectable radiographically. He found that overlying radiopaque structures obscurred the experimentally produced lesions. Also, simulation of soft tissue replacement of bone following removal caused only a slight porosity on the radiograph. He concluded that this porosity would unlikely be noticed in the intact patient. Working with cadaver vertebrae, he realized early that quite large lesions might go undetected. The work of Schackman and Harrison[14] confirmed this finding. They were able to demonstrate microscopically visible metastasis which were radiographically invisible while working on vertebrae and long bones. Goldman, Millsap, and Brenman[15] discovered only a slight density change on the radiograph when buccal and lingual cortical plates were removed from cadaver specimens. They also found no change in the lamina dura radiographically when the cortical plates were removed. The periodontal ligament space and trabecular pattern remained intact. Therefore, they concluded that when a radiolucent area was seen on a radiograph, the bucco-lingual extent of the area could not be determined nor could it be determined if one or both of the cortical plates were involved. In their opinion, a part of the cortical plate might have been missing without any apparent change in the radiograph because the trabecular pattern of the spongy bone

masked its appearance on the radiograph. Also, registration of the lamina dura resulted from the presence of dense bone extending, for all practical purposes, the entire buccal-lingual extent of the tooth. The quantity, therefore, and not the nature of the bone, determined its appearance on the radiograph.

According to Sicher[16], the mandible is made up of cortical and cancellous bone. The alveolus and lamina dura are cortical bone and are continuous with the outer buccal and lingual cortical plates of the body of the mandible. He also stated that cancellous bone is relatively radiolucent and hence less dense than the cortical bones which surround it. The cortical bone is dense because of its lower content of fibrillar matter and higher content of cementing substance. The cementing substance contains a greater content of calcium salt per unit volume. With respect to this information, the first of two articles by Bender and Seltzer[17] in 1961 gave startling results. Bony lesions could not be detected radiographically unless a cortical plate was perforated, gross destruction of outer cortex occurred, or erosion of inner cortical plate took place. Those lesions confined completely within cancellous bone could not be visualized on conventional dental radiographs. What was thought to have been cancellous bone destruction (viewed as loss of trabecular pattern) was actually erosion of the innermost cortical surface at the area of the junction between cancellous and cortical bone. The defect could not be visualized beyond the junctional area as it encroached upon marrow spaces. Therefore, they reported that large lesions confined to cancellous bone could go completely undetected by the

radiograph. Whereas this first study involved gross removal of bone, a follow-up study in the same year by Bender and Seltzer was more specific in nature.[18] Having produced experimental lesions of periapical and periodontal structures, they found once again that lesions entirely within cancellous bone were undetectable. Only if the inner cortex was eroded or if the junctional area between cancellous and cortical bone was affected did the area show up radiographically. In this study, the position of the root apex in relation to the cortical plate was described. Since, according to Bender and Seltzer, apices of most teeth are lodged in or near cortical bone, then development of periapical lesions appears relatively quickly. Most roots were described as in close proximity to the buccal cortical plate save for the mandibular third molar. Other mandibular molars, according to Bender and Seltzer, were more likely entirely within cancellous bone. It was also noted that the position of the lesion (buccal or lingual) could not be detected.

While these authors compared gross appearance of lesions to radiographic evidence, Schwartz[19] compared preoperative versus postoperative radiographs to determine possible evidence of radiographic changes. Although his experimentally produced lesions also penetrated cortical plate or lamina dura, this destruction of cortical bone was not observed radiographically. He reported superimposition of buccal and lingual cortical plates over the induced alveolar crest defect as the obscurring factor on the radiograph. In most of the defects produced in this study, only cancellous bone was destroyed. In spite of extensive destruction, the cavitation was recorded only as a change in the comparative density

of the overall area radiographed. Removal of trabeculae allowed for greater penetration of the x-ray beam resulting only in a darker image in general.

In an evaluation of naturally occurring periapical radiolucencies in cadavers, Regan and Mitchell[20] found that only four of eighteen lesions appearing radiographically did not perforate one of the cortical plates. Of those in which perforation did not occur, severe erosion of one of the cortical plates did occur as well as removal of junctional trabeculae. Junctional trabeculae were defined to be "...those ridges on the endosteal surfaces of bone which remain after the central or marrow trabeculae are removed." Garber[21] supported these results by showing that an area of rarefaction seen on a radiograph depends upon the thickness of over-lying bone and the amount of destruction of the cortical plate. He added, however, that although the lower teeth showed a greater tendency for radiolucencies to appear than maxillary teeth, the lower molars do not fit this pattern. Ramadan and Mitchell[22] reported similar results. They studied many types of experimentally produced lesions of bone on one skull and mandible. They found that central bone trabeculae destruction did not show up on the radiograph unless junctional trabeculae were also destroyed. Combined with cortical plate removal, such destruction became much more apparent. Like Goldman and associates[15] however, it was found that removal of the entire buccal or lingual cortical plate did not markedly alter the radiograph. Ramadan and Mitchell explained that bone loss was obscurred by the shadow of the roots which were more dense than the cortical plates.

According to Wengraf[23], dried bones gave far greater contrast radiographically than did bones *in vivo*. He also stated that it was common to find the actual lesion much larger than the radiographic size of the lesion as evidenced during surgical procedures. He stated that a lesion was only dependent upon cortical erosion for radiographic appearance. Furthermore, the relationship of the apices to the cortical bone might cause a lesion to show up very easily.

Pauls and Trott[24] confirmed the work of Bender and Seltzer[17,18] and Wengraf.[23] They too concluded that the reason so many lesions were seen radiographically was due to the relationship of the apex of the root to the inner aspect of the cortex of the mandible. In the human mandible the anterior and bicuspid teeth have their apices near the buccal aspect of the inner surface of the cortical bone. Pauls and Trott also stated that the mandibular first molar has its mesial root near the buccal plate while the distal root is either centrally placed in cancellous bone or near the lingual cortical bone. The mandibular second molars usually have their apices near the lingual cortical plate. Like Wengraf[23], they found that the actual size of a lesion during surgery was always greater than the extent to which the radiograph would indicate. Assimilation of these facts brought the authors to conclude that early radiographic change was not synonymous with early or insignificant pathological change.

Orban[25] and Elfenbaum[26] believed that the lamina dura was more highly calcified than other cortical bone. Therefore, any break in continuity of the lamina dura was evidence of a developing disease process. In opposition, Goldman, Millsap, and Brenman[15], and Manson[27], showed that the angle of the radiograph was an important factor, that

the density of cortical bone does not vary in different parts of the mandible, and variations in the appearance of the lamina dura should not determine the diagnosis of periodontal or periapical disease. In 1970, Van der Linden[28] showed that the visual subjective width of the periodontal ligament space might be altered by a number of technical factors.

Rees and associates[29], studying cadaver specimens, found that proximal osseous defects on the buccal or lingual aspects of multirooted teeth could be identified with a high degree of success from their radiographic appearance. However, those lesions presenting on the buccal or lingual surfaces of the alveolar arch were extremely difficult to locate. They concluded that the more easily visualized lesions presented because they were naturally occurring in cadaver specimens versus experimentally produced lesions which might not have been as easily visualized. These results are in agreement with those of other authors.[17,18,22]

Phillips and Showkat[30] demonstrated that, in spite of cortical plate involvement, defects produced in dried specimens consistently showed up better on panoramic radiographs than on conventional radiographs due to the tomographic principle innate to panoramic radiography.

Volchansky and Cleaton-Jones[31] recently studied bony defects on dried Bantu mandibles. Although few periapical lesions were detected radiographically, more were detected radiographically than were visually apparent. Perforation of the buccal cortical plate was the indicator used as visual evidence of a periapical lesion.

In contrast to the preceding findings, only a few investigators have found cortical plate involvement unnecessary in the ability to observe periapical lesions radiographically. Shoha, Dowson and Richards[32]

used an exposure time of two seconds (55 KVP and 10 Ma) and found that, indeed, the size of a bony lesion was larger than its radiographic image although only slightly so in the premolar region of a dried specimen. In this specific area, neither junctional nor cortical bone were found to be involved. In agreement, Smith[33] reported that when dealing with a subacute or chronic stage of infection within the bone, once the inflammatory process has passed through the lamina dura, the infection would easily spread into the trabecular bone surrounding the tooth. In Smith's opinion, at this point the radiographic appearance was that of a radiolucent area associated with the apex of the tooth. Because no special features exist to prevent uniform spread, the lesion proper was approximately circular. Radiographically, however, the outline of the radiolucent area was often indefinite. Only if left untreated at this time, according to Smith, would the cortical plates be breached.

LeQuire, et al.[34], investigated artificially created lesions in the cancellous bone of dry human mandibles. Using 90 KVP, 15 Ma, and 0.7 or 0.8 second and stringent radiographic technique, these authors found that in a very high percentage of cases, lesions produced in cancellous bone alone are radiographically visible. They concluded that this was due to the quality of the x-ray beam used and the wider gray scale offered by the use of higher voltage.

C. USE OF THE DOG AS AN EXPERIMENTAL MODEL

Compounding the criteria of Tagger[35] and Scoralle[36], the criteria for choosing an ideal test animal are as follows:

1. Morphologically the teeth should be similar to the human.
2. The periapical tissues should resemble those of the human anatomically and histologically.

3. The susceptibility to periodontal infection should be comparable to the human.
4. The inflammatory response should be applicable and consistent.
5. The animal must not be allergic to any implanted materials.
6. The external and internal tooth dimensions ought to approximate those of a human.
7. Routine dental armamentarium should be adaptable to the animal.
8. The animal must be able to withstand the procedures.
9. The diet must be controllable.
10. The availability, cost, and care of the animal should be reasonable.

Scoralle concluded that the dog was an acceptable model for an *in vivo* comparison of endodontic filling materials. He did not study periapical lesions. At any rate, he favored the mandibular premolar of the dog for use because of its relative ease in being radiographed. Due to the flat palate of the dog, exaggerated angulations necessitate distorted images of the maxillary teeth. This author also described the dental formula for the dogs' normal dentition (permanent). It totals 42 teeth and reads as follows: 2(I 3/3, C 1/1, P 4/4, M 2/3). He further reported that brachycephalic (short-headed) dogs might have a decreased number of molar teeth. Carnassial (sectorial) is a term frequently applied to the maxillary fourth premolars and mandibular first molars. He reported that the mandibular last premolar and first molar were two-rooted teeth. Also, Scoralle indicated that from first premolar through first molar, the roots became progressively longer. In his study, the root canals of all premolars studied were the same morphologically.

That is to say, they were all straight and without significant taper as one approaches the apex. The pulp was found to exit through multiple tiny foramina, usually four or more. Because extraction of these teeth was difficult, the author described some possibilities to explain this problem. He stated that the attachment apparatus might be more tenacious than in man, the bone might be histochemically more dense, there appeared to be only a modest amount of cancellous bone between the cortical plates, and the lamina dura might be broader around these roots. All or any of those statements, if true, could make extraction more difficult. He did not discuss divergent roots as a possible cause for difficulty in extraction or extremely thick and dense cortical plates.

In 1913, Grove[37] utilized the dog as an experimental model, observing the histologic effects of formaldehyde on periapical tissues. In 1916, he[37] again utilized the dog in testing radiographic and histologic reaction to certain root canal medicaments. He observed severe inflammatory changes and abcesses in the periapical tissues as a result of using formocreosol in both studies. In 1932, Coolidge[39] used dogs and found that fifteen different medicaments used in endodontic therapy were irritating upon histologic analysis. He realized the possible problems in relating one experimental model to another but concluded that results from this model were applicable to human tissue responses. Orban[40] repeated a similar experiment and agreed with the results of Coolidge.[39] Also in 1932, Hill[41] described the experimental production and histology of dental granulomas in dogs. He concluded that streptococcus was the bacteria of choice to produce pulpo-periapical lesions in dogs and because multiple apical foramina exist in the dog tooth, it might be easier to infect dog teeth than human teeth. He found resorption

of the root surface commonplace and eipithelial proliferation normal after three months following infection of the pulp with streptococci. Resorption of the external root surface when a lesion was present was representative and even in normal control teeth, apposition of new cementum over old cementum continued as a normal aging process.

Dixon and Rickert[42] also concluded that tissue responses in the periapical region of the dog and human were identical and the histologic response was comparable to man.

However, in 1971, Barker and Lockett[43] related that canine pulpal tissue might not be as resistant to pulp capping procedures as their human counterparts.

Lawson, et al.[44], in 1960, reviewed the dental anatomy of the dog. In addition, these authors covered the histology of dog enamel, dentin, cementum, pulp, and periodontal ligament. No comparison was made to the human counterparts.

Another study was done in 1969 concerning the reaction of alveolar bone and cementum to experimental abcess formation in the dog. In this study[45], Torneck and Tulananda found that due to the normal periapical thickening displayed by the dog, any radiographic investigation provided only an approximation of the bone pathoses in the area. Also, they reported that the apical one-third of the roots studied were covered by a relatively thick layer of cellular cementum related to secondary eruption which was normal in the dog. Where inflammatory periapical lesions were present there existed increased deposition of bone about the periphery of the remaining trabeculae except when inflammatory cell accumulation was intense. In this instance, osteoclasts and resultant bone resorption were evident. Also, it was observed that in teeth which had large

periapical radiolucencies, the "osseous side" of the periodontal membrane space displayed osteogenic activity especially on the buccal aspect. Where periapical lesions were seen and substantial bone resorption occurred, bone deposition was seen throughout the remainder of the periodontal space. Whatever the cause, this reaction represented a periodontal response to a periapical lesion. All periapical lesions of relatively long standing (i.e., more than 83 days) demonstrated the deposition of new endosteal bone on the side away from the lesion or along the sides of trabeculae approximating the lesion. In contrast, those areas in which intense inflammation or pus formation were evident showed resorption of bone rather than its deposition. According to this author, bone resorption and deposition patterns seen in the dog might be unique to the dog and incomparable to other models.

D. EXPERIMENTAL PRODUCTION OF PULPO-PERIAPICAL LESIONS *IN VIVO*

In 1966, Grossman[46] found that certain strains of microorganisms required only one organism to initiate growth in a culture medium. He found that certain culture media were preferred over others and that different strains within the same species of bacteria grew better in one medium than in another. Some strains of Streptococcus faecalis which were tested grew out with only one organism originally present in culture. Palmer, Lazzarotto, and Weine[47] showed that as few as three organisms were necessary to initiate growth in a culture medium. In 1930, Dubos[48] stated that the ideal culture medium should support the growth of a single microorganism.

According to Torneck[49], the relationship of microorganisms to endodontic disease was twofold: a) The microorganisms might establish

disease within the pulp and periapex (primary effect), and b) Microorganisms might prevent healing from occurring (secondary effect). He stated that the role of the microorganism in producing periapical disease was best described as being passive, for they were unable to directly infect the surrounding tissues. Only under specific circumstances could this situation be altered: a) Superimposition of a systemic disturbance, b) Predisposing local injury, c) A local increase in the number and growth of certain virulent microorganisms, or d) A symbiotic effect of two or more groups of microorganisms. In these circumstances, periapical infection could occur and the role of the microorganism was considered active. Torneck also implied that the substrate necessary for microbial growth was present in the root canal. Moreover, he warned of the problems involved in directly relating animal experiments to man. Animals were not always susceptible to the same microorganisms pathogenic to man, nor did animals always respond immunologically in the same manner as did man.

Other problems might occur. Garcia, Jansons, and Kapur[50] did a study in which they attempted to detect periapical pathology in the dog. They found in general that periapical pathology of infectious origin was found to stimulate an increase rather than a decrease of osseous activity in the form of bone deposition. They also related the fact that the dog molar had a wide periodontal membrane space which was normal and radiolucent under most conditions. These authors indicated four to six weeks as the shortest time period possible in order to produce periapical radiolucencies. However, after two weeks a slight pathological widening of the periodontal membrane space and loss of lamina dura registration was observed immediately adjacent to the apex.

In a study of 4,000 root canal cultures of humans, Winkler and Van Amerongen[51] pointed to Streptococcus faecalis as the major problem organism in human root canal therapy. As a group, streptococci persisted tenaciously in the root canal system. Subgroup liquefaciens was particularly pathogenic.

Kasle and Klein[52] septically and aseptically produced lesions in the periapical tissues of dogs. They simply left vital pulps exposed to the normal oral flora present in oral fluids. Aseptically, they produced lesions by placing a saturated pledgett of ammonium hydroxide into the pulp chamber. The canal was then sealed from oral contamination. Their first suggestive evidence of periapical pathosis was noted at 22 days and not confirmed until 29 days. Torneck and Tulananda[45] left dogs' teeth exposed to the oral environment in order to propagate lesions. Barker and Lockett[43] utilized Streptococcus viridans (an alpha hemolytic streptococcus) to infect dogs' teeth. This bacteria was said to incite a chronic to subacute periapical response without danger of acute exacerbation. According to this study, the periapical lesion became radiographically visible within two to three months.

E. EXPERIMENTAL TECHNIQUE FACTORS

Using the dog as an experimental model, Kasle and Klein[52] utilized a custom headrest for their beagles. In so doing they were able to produce consistent periapical radiographs in a serial fashion. Constant distance of cone to object was accomplished using a rod attached to individual intra-oral compound impressions for each dog. In this method, reproducible technical factors led to reproducible anatomic structures on serial radiographs. These authors were attempting to radiograph

early periapical radiolucencies, hence the need for reproducible radiographs from one experimental period to the next. Scoralle[36], on the other hand, held the x-ray packets with a hemostat in the areas which he radiographed. Duinkerke, et al.[53], used a paralleling device which fit intraorally and extended extraorally to allow standardized projections on the dental radiograph. He claimed that projection error was negligible with this system. Duinkerke also gave instructions concerning kilovoltage peak, milliamperage, and time of exposure necessary to radiograph the dog and obtain satisfactory radiographs. He used 90 KVP at 10 Ma for 0.5 second on a portable hospital x-ray unit by adding a cone to the collimator. Ramy and Segretto[54] used 62-70 KVP, 100 Ma, and 0.2 second with a 36 inch object to target distance. McCormick[55] used 60 KVP, 20 Ma, 0.2 second, and a distance of 20 centimeters. Obviously, acceptable radiographs were a function of the x-ray machine being used in the experiment.

Once the radiographs had been prepared, it remained to compare successive examples. Duinkerke and van de Poel[56], using an exacting measurement technique, determined that although two radiographs of the same object appeared to be identical when lying side by side, they could differ considerably. Therefore, limitations in assessment of diameter changes of periapical radiolucencies were observed. For example, positioning error (angulation of the x-ray beam through the lesion) for maxillary molars of humans was determined to be a factor of 0.83 millimeters. This standard deviation means that a radiolucent area in this location measured to be four millimeters on the radiograph could be in actuality between 1.5 millimeters and 6.5 millimeters in size. Because of projection differences, it was impossible to assume the angulation

and hence size of a lesion on two apparently identical radiographs. In a separate study, Duinkerke and associates[57] determined that comparisons of two separate interpretations of measurements of periapical radiolucent areas resulted in an average error of twenty-one per cent in well defined lesions and thirty-seven per cent for diffuse lesions. Like Sommer, Ostrander, and Crowley[58], a common method used to measure a lesion on the radiograph was a millimeter measurement of the greatest radiolucent diameter.

CHAPTER III

MATERIALS AND METHODS

This project was initiated with the intention of comparing the visual, radiographic, and histologic properties of pulpo-periapical lesions in the dog. The reasons for finding and using an acceptable model for this type of research are derived from an understandable lack of human subjects and the unreasonable cost of primate research. The availability, reasonable cost, and easy maintenance of the Beagle dog are among the reasons it was chosen for use in this experiment. As indicated earlier, other authors[38,42,44] had found the dog acceptable for endodontic research. One important reason for their choice was the relative accessibility of the mandible and tooth size, which are comparable to the human.

In this study five adult Beagle dogs were used. The dogs were procured through the Animal Research Facility at the Loyola University Medical Center. After their arrival at the Research Facility, the dogs were observed for a minimum of two weeks to ensure health and adaptation to a new environment. One dog (#17) expired during this observation period. The remaining four animals weighed between 6.5 and 10.5 kilograms (kg). Each dog was identified by a numbered collar tag. This number was recorded on all experimental data collected in order to categorize the results. The dogs were not fed on the scheduled operative day to avoid complications during maintenance of general anesthesia.

General anesthesia was always administered by intravenous injection of one cubic centimeter (cc) of sodium pentobarbitol* for each two kg. of body weight. According to the manufacturer, one cc contained 65 milligrams (mg) of this long acting barbiturate. The anesthetic solution was injected into a superficial vein which is accessible on the medial aspect of the shaved forearm of the dog. Induction of initial anesthesia was uncomplicated in all cases. The dog was then secured to an operating table with tape to facilitate operative or radiographic procedures.

During the first operative procedure, and due to its unusual length of time, supplemental anesthesic doses were necessary for all experimental animals. Dog #16 regained conciousness very quickly during the operative procedure. In its agitated and/or excited state, intravenous re-injection was made difficult due to the return of normal reflexes. After several attempts, intravenous injection of at least one cc of anesthetic solution was accomplished. This dog subsequently expired of apparent anesthetic overdose, leaving only three dogs for continued experimental purposes.

The maxillary and mandibular jaws were retracted using a spring-loaded device which attached to the cuspid teeth on the side opposite that being operated. The fourth premolar and first molar in each quadrant were chosen for experimental procedures. Therefore, eight teeth in each of the three remaining dogs were operated and radiographed on the first day of experimental procedures.

The teeth were isolated by buccal and lingual placement of four by four inch non-sterile gauze sponges. The teeth were swabbed with a

---

*(W. A. Butler Co., Columbus, Ohio)

seventy per cent alcohol solution and an opening was made into the pulp chamber using heatless stones and straight fissure burs mounted on standard low speed and high speed handpieces, respectively. All instruments used were sterilized and/or disinfected prior to use. The access cavity openings were then sealed carefully with silver amalgam.*

Next, a device was fabricated for each quadrant of each animal to facilitate the taking of superimposable serial radiographs. Acrylic tray plastic** was mixed and molded around the teeth of the individual dog as well as a wooden tongue blade (Fig. 1) so that angulation of cone to target was standardized and error minimized. The acrylic "impressions" were trimmed after they had set so that trauma to the soft tissues of each animal was prevented.

Utilizing these devices, the first (and all subsequent) radiographs were taken on the same dental x-ray machine.*** All radiographs taken throughout the experiment were subsequently processed in the same automatic processor****; utilizing the same technique factors for consistent quality each time. Kilivoltage peak and milliamperage-seconds were varied until satisfactory films were acquired with respect to density and contrast. The radiographs which were taken immediately following the operative procedure were used as controls, as were those of a block section of mandible taken from an unoperated dog obtained at a later date. The animals were then returned to their cages at the Animal Research

---

*Tytin - S. S. White Co., Great Neck, New York

**Coe Tray Plastic - Coe Laboratories, Chicago, Illinois

***Flexomatic 90 - S. S. White Co., Great Neck, New York

****Philips 810 - Philips Dental Division, Stamford, Connecticut

Facility as they were following each subsequent experimental period and maintained on a soft diet until the time of sacrifice. Altogether, there were nine experimental days.

Any remaining coronal pulp tissue was removed with an endodontic excavator. Access cavities were then enlarged in order to facilitate experimental infection of these teeth. Each canal orifice was located with an endodontic explorer. Standardized K-type endodontic files* were then used to macerate the contents of the canals to the depth of the apical delta. The canals were irrigated with sterile saline to remove gross blood and debris and dried with sterile paper points.

A standardized amount of a previously prepared pure culture of Streptococcus faecalis** was deposited into the enlarged access cavity preparations. The S. faecalis used (an alpha-hemolytic enterococcus, group D) was grown for 18 hours at $37^{o}C$ in Brain/Heart Infusion broth. This suspension was then centrifuged at fifteen thousand gravities (g) for twenty minutes and washed twice with saline. After each wash it was resuspended to the original broth culture volume with saline. The organisms were then diluted five-fold to yield 4.6×10^{6} organisms/25 µl volume. A standard 25 µl micropipette was used to innoculate each experimental tooth with the specified number of microorganisms. The solution was deposited over a small sterile cotton pellet which was placed in each pulp chamber. In every case, the dog was positioned so that gravity aided the placement of the suspension.

---

*Union Broach Company, Inc., Long Island City, New York

**Midwest Cultures, Terre Haute, Indiana

All three dogs were sacrificed on the 44th day when all experimental teeth were observed to exhibit frank pulpo-periapical radiolucencies on the corresponding periapical radiographs. The dogs were sacrificed by intracardiac puncture and subsequent injection of Beuthanasia-D Regular.* The active ingredients of this preparation are pentobarbital sodium (196 mg/ml) and phenytoin sodium (25 mg/ml). The recommended dosage is one ml/four kg. Block sections of maxillae and mandibles containing the experimental teeth were removed immediately and placed in ten per cent buffered neutral formalin.

The mandibular teeth and surrounding tissues were chosen for subsequent histologic sectioning and analysis. The sections containing the roots of the mandibular fourth premolar and first molar were sectioned in a buccal-lingual direction (Fig. 2) to separate each root into an individual specimen of tooth, pulpo-periapical lesion, and bone. One block section of mandible containing the experimental teeth was sectioned in a mesio-distal direction (Fig. 3). The specimen utilized was the mandibular left block section from dog #19. This was accomplished in order to visualize better the existing pulpo-periapical lesions from the same viewpoint as the periapical radiograph. The apical one-third of the roots of the experimental teeth were carefully removed using a mounted green stone on a low speed handpiece. The purpose of this exercise was to allow better viewing of the pathosis. All specimens were then placed in separate bottles of formalin solution and labelled in order to identify each one throughout the remainder of the experiment. Each specimen was marked to indicate the mesial aspect of the block for

---

*Burns - Biotec Laboratory, Oakland, California

histologic sectioning and counting purposes. Each bottle was marked with a code which identified the segment as follows:

Example Label: #22-LR-PRE-DRT

#22 = dog collar tag
LR = mandibular right
PRE = fourth premolar
DRT = distal root

Each specimen was then radiographed from the buccal and from the mesial. The formalin solution was replaced three times during the two weeks of fixation prior to decalcification.

All specimens were then decalcified in D-Calcifier*, a pre-packaged histological decalcifier. The active ingredient in this solution is hydrochloric acid (44.4 percent/weight). They were they dehydrated in alcohol, cleared in xylol and paraffinated in an auto-technician.** The specimens were imbedded (mesial aspect exposed) in paraffin and five micron sections were taken at 200 micron intervals throughout the specimen. Each section was placed on a single glass slide. The sections were fixed on the slides with egg albumin, the paraffin was melted off and the tissues dried in an oven heated to 60°F. They were then stained with hematoxylin and eosin in the usual manner by a technician in the Oral and General Pathology Department at Loyola University School of Dentistry.

Radiographs of the specimens were analyzed with respect to the presence of pulpo-periapical lesions. The radiographs of lesions in the live dog as well as those of each specimen were measured with a millimeter ruler and compared. The histologic sections were counted in order

---

*Lerner Labs, Stamford, Connecticut

**Technician Co., Chauncey, New York

to attempt to determine the cellular beginning and ending (width) of each pulpo-periapical lesion. Data was collected concerning the size of each lesion, its appearance grossly as well as histologically, and the position of the root apex with respect to its associated lesion and the cortical plates. An attempt was also made to account for any differences which occurred in the size of each lesion radiographically and histologically. From the histologic sections, the predominant features of an existing pulpo-periapical lesion were assessed.

CHAPTER IV

RESULTS

The Beagle dog was utilized as an experimental model in this study. A radiographic and histologic comparison was made of pulpo-periapical lesions induced in mandibular teeth of the dog. An attempt was made to describe the normal architecture as well as pathoses surrounding the roots of the fourth premolar and first molar. The radiographic portion of the study was limited to size and shape comparisons. Detailed analysis of the structures involved in pulpo-periapical lesions as well as size and shape comparisons were obtained from the histologic sections.

As was mentioned earlier, two dogs expired early in this experiment. The remaining three dogs tolerated all procedures well. At the time of sacrifice none had lost weight or appeared debilitated.

The detailed description of a pulpo-periapical lesion is a difficult task, since of necessity, it is based upon the subjective interpretations of the viewer. In order to categorize the important findings, the data was arranged in tables related to the following parameters:

(1) The comparative size of the lesions both radiographically and histologically;

(2) The position of the root within the mandible;

(3) The distribution of the lesions around their respective root apices;

(4) The associated structures affected by or involved with the lesions.

The information gathered was intended to direct the research of others with respect to the applicability of results to the human counterpart.

NORMAL FINDINGS -- Both the mesial and distal roots of the mandibular first molar of the Beagle dog exhibited wide periodontal membrane spaces at their apices. This finding was observed radiographically and histologically (Figs. 4 and 5). The apical segments of all roots were surrounded by a layer of secondary cementum thickest at the apex as determined by histologic analysis. Clinically and radiographically, the main canal of each dog's tooth ended abruptly in the apical portion. This was determined by the placement of standard K-type endodontic files into the canal until they would go no further (Fig. 6). At this point the canal branched into several smaller canals, or an apical "delta-like" configuration (Fig. 7).

The general architecture of the dental complex of this animal model, including the mandible, was similar to the human counterpart. Dentin, cementum, periodontal membrane, cortical and medullary bone, marrow spaces and a mandibular canal were always observed.

Specifically, the bony architecture of the dog mandible and the position of the root apices with respect to the mandibular canal were interesting. The buccal and lingual cortical plates were found to be extremely thick and dense to the extent that very little medullary bone existed. That medullary bone which was present seemed to be composed of thick, dense spicules which allowed for a limited number and amount of fatty marrow spaces (Fig. 8). With the entire length of a root present in any histologic section, there was little room in any direction for

medullary bone or marrow spaces between the cortical plates of bone. In all specimens viewed, there existed a limited component of bone separating the mandibular canal from its adjacent structures above (Fig. 9). The mandibular canal seemed to be composed of the same major components as the human mandibular canal. That is to say, a large nerve, artery, and vein, surrounded by connective tissue were always observed.

PATHOLOGIC FINDINGS -- The roots of all experimentally infected teeth exhibited the presence of pulpo-periapical lesions. Each exhibited communication through, disruption of, or complete absence of the bony demarcation between the mandibular canal and adjacent structures above (Figs. 10, 11, and 12). Bone resorption in the area of the lesion as well as bone apposition were commonly observed. Root resorption was also a frequent finding, especially in the central portion of the lesion. A necrotic center, mixed (fibrous connective tissue present) infiltrate and a purely cellular infiltrate into the area of the mandibular canal were also common observations (Tables I and II). Bone resorption as well as root end resorption appeared jagged, while normal or newly formed bone appeared smooth and exhibited gentle curves (Figs. 13 and 14).

Pulpo-periapical lesions consisted of a central area of necrosis surrounded by varying degrees of inflammatory cell infiltrate and fibrous connective tissue. The fibrous connective tissue-cellular infiltrate complex partially encapsulated the necrotic portion of the lesions. The lesions appeared largest in diameter (viewed from the mesial) where frank necrosis existed histologically. The different pathologic processes constituting the lesion were visually observed to dimish in number and intensity as the outer limits of each lesion were reached (Appendix

TABLE I

RELATIVE HISTOPATHOLOGIC FEATURES APPARENT IN THE MIDDLE 1/3 OF LESION

Specimen #	Dog #	Side	Tooth	Root	Necrosis	Cellular Infiltrate Only	Mixed (Fibrous Tissue Present)	Bone Resorption	Bone Apposition
C	Control	LR	PRE	MRT	0	0	0	0	0
4	#22	LR	PRE	MRT	0	M. Canal	++	+	+
16	#14	LR	PRE	MRT	+	M. Canal	++	+	+
19	#19	LR	PRE	MRT	+	M. Canal	+	+	+
9	#22	LL	PRE	MRT	0	M. Canal	++	+	+ (M. Canal only)
11	#14	LL	PRE	MRT	+	M. Canal	+	+	+
D	Control	LR	PRE	DRT	0	0	0	0	0
3	#22	LR	PRE	DRT	0	M. Canal	+	+	+
17	#14	LR	PRE	DRT	0	M. Canal	++	+	+
18	#19	LR	PRE	DRT	+	M. Canal (++)	0	+	+
8	#22	LL	PRE	DRT	+	M. Canal	+	+	0
10	#14	LL	PRE	DRT	+	M. Canal	++	+	+
B	Control	LR	MOL	MRT	0	0	0	0	0
5	#22	LR	MOL	MRT	+	M. Canal (++)	0	++	+ (M. Canal only)
14	#14	LR	MOL	MRT	+	M. Canal	++	+	0
20	#19	LR	MOL	MRT	+	M. Canal (+)	0	++	+
7	#22	LL	MOL	MRT	+	M. Canal	++	+	0
13	#14	LL	MOL	MRT	+	M. Canal	++	+	0
15	#14	LR	MOL	DRT	+	M. Canal	++	+	0
21	#19	LR	MOL	DRT	+	M. Canal	0	+	+
6	#22	LL	MOL	DRT	+	M. Canal	++	+	+
12	#14	LL	MOL	DRT	0	M. Canal	++	+	?

TABLE II

RELATIVE HISTOPATHOLOGIC FEATURES APPARENT IN THE OUTER 1/3 OF LESION

Specimen #	Dog #	Side	Tooth	Root	Necrosis	Cellular Infiltrate Only	Mixed (Fibrous Tissue Present)	Bone Resorption	Bone Apposition
C	Control	LR	PRE	MRT	0	0	0	0	0
4	#22	LR	PRE	MRT	+	M. Canal	+	+	+
16	#14	LR	PRE	MRT	++	M. Canal	++	+	0
19	#19	LR	PRE	MRT	+	M. Canal	++	+	+
9	#22	LL	PRE	MRT	+	M. Canal	++	+	+
11	#14	LL	PRE	MRT	+	M. Canal	++	?	?
D	Control	LR	PRE	DRT	0	0	0	0	0
3	#22	LR	PRE	DRT	-	M. Canal	+	+	0
17	#14	LR	PRE	DRT	+	0	+	+	0
18	#19	LR	PRE	DRT	0	?	+	+	+ (M. Canal only)
8	#22	LL	PRE	DRT	0	M. Canal	++	?	+
10	#14	LL	PRE	DRT	0	M. Canal	+	0	0
B	Control	LR	MOL	MRT	0	0	0	0	0
5	#22	LR	MOL	MRT	++	0	++	++	+
14	#14	LR	MOL	MRT	+	M. Canal	+	++	+
20	#19	LR	MOL	MRT	0	M. Canal	++	++	+
7	#22	LL	MOL	MRT	++	M. Canal	++	+	+
13	#14	LL	MOL	MRT	+	M. Canal	++	++	+
15	#14	LR	MOL	DRT	0	0	++	?	+
21	#19	LR	MOL	DRT	+	M. Canal	+	+	+
6	#22	LL	MOL	DRT	+	0	++	+	+
12	#14	LL	MOL	DRT	0	M. Canal	++	+	+

Tables VIII-XVII). The tissues determined to be affected in the presence of a pulpo-periapical lesion are indicated in the legend for these tables. The analysis of these tables indicated that fewer changes were affected as the outer limits of the lesion were reached. In most cases, the central area of the lesion corresponded to both buccal and lingual cortical plate involvement. Accordingly, resorption of medullary bone and cellular infiltrate into the mandibular canal accompanied disruption of both cortical plates of bone. Cellular infiltrate of the mandibular canal was a commonplace finding throughout the histologic sections of each lesion. The section from the approximate center of the root apex and root canal space consistently corresponded to the area of greatest destruction.

The position of the root apex with respect to the cortical plates of bone was indicated in Figs. 15, 16, 17, and 18, and in Table III. There is a shift from buccal position to lingual position of the root apex from mesial root of fourth premolar to distal root of first molar. Nearly all of the molar roots are positioned lingually, but especially the distal root of the first molar.

The position of the individual root apex with respect to the cortical plates of bone also seemed to dictate the direction in which each lesion progressed. Table IV was developed to indicate the distribution of the pulpo-periapical lesion around each infected root. The major factors which influenced the spread of the lesions were the position of the root apices to the cortical plates of bone and mandibular canals. Radiographs taken from the buccal and from the mesial, as well as buccal-lingual histologic sections, provided information concerning the three-dimensional spread of these lesions.

TABLE III

RELATIVE POSITION OF ROOT APEX TO CORTICAL PLATES OF BONE

(HISTOLOGIC SECTIONS VIEWED FROM THE MESIAL)

Specimen #	Dog #	Side	Tooth	Root	Buccal	Central	Lingual
C	Control	LR	PRE	MRT	+		
4	#22	LR	PRE	MRT	+		
16	#14	LR	PRE	MRT	+		
19	#19	LR	PRE	MRT		+	
9	#22	LL	PRE	MRT	++		
11	#14	LL	PRE	MRT	+		
D	Control	LR	PRE	DRT		+	
3	#22	LR	PRE	DRT	+		
17	#14	LR	PRE	DRT		+	
18	#19	LR	PRE	DRT		+	
8	#22	LL	PRE	DRT		+	
10	#14	LL	PRE	DRT	+		
B	Control	LR	MOL	MRT			+
5	#22	LR	MOL	MRT			+
14	#14	LR	MOL	MRT			+
20	#19	LR	MOL	MRT			++
7	#22	LL	MOL	MRT		+	
13	#14	LL	MOL	MRT			+
15	#14	LR	MOL	DRT			++
21	#19	LR	MOL	DRT			++
6	#22	LL	MOL	DRT			++
12	#14	LL	MOL	DRT			++

++ = Extremely Close or Touching Respective Cortical Plate

TABLE IV

RELATIVE DISTRIBUTION OF PULPO-PERIAPICAL LESION TO ROOT APEX

					A. Histologic Sections Viewed from Mesial			B Radiographs from Buccal			C. Radiographs From Mesial		
Specimen #	Dog #	Side	Tooth	Root	Buccal	Apical	Lingual	Buccal	Apical	Lingual	Mesial	Apical	Distal
C	Control	LR	PRE	MRT	- No Lesion Present -			- No Lesion Present -			- No Lesion Present -		
4	#22	LR	PRE	MRT	+	++	++	+	+	++	++	++	++
16	#14	LR	PRE	MRT	+	++	++	0	++	+	++	++	+
19	#19	LR	PRE	MRT	+	+	++	0	+	++	0	+	++
9	#22	LL	PRE	MRT	+	+	++	+	+++	++	++	++	++
11	#14	LL	PRE	MRT	+	++	+	0	0	+++	++	+	++
D	Control	LR	PRE	DRT	- No Lesion Present -			- No Lesion Present -			- No Lesion Present -		
3	#22	LR	PRE	DRT	+	++	++	+	++	+	++	++	++
17	#14	LR	PRE	DRT	++	++	++	+	+++	+	++	++	++
18	#19	LR	PRE	DRT	+	++	++	+	++	0	+	+	++
8	#22	LL	PRE	DRT	++	++	++	+	++	+	++	++	+
10	#14	LL	PRE	DRT	++	++	++	++	++	++	++	++	++
B	Control	LR	MOL	MRT	- No Lesion Present -			- No Lesion Present -			- No Lesion Present -		
5	#22	LR	MOL	MRT	0	+	+++	0	0	++	+	++	+++
14	#14	LR	MOL	MRT	++	++	++	+	++	+++	++	++	++
20	#19	LR	MOL	MRT	++	+	+	0	++	++	++	+	++
7	#22	LL	MOL	MRT	0	++	++	0	+	+++	+	+	+++
13	#14	LL	MOL	MRT	+	+	++	+	0	+++	+	++	+++
15	#14	LR	MOL	DRT	+	++	++	0	++	++	+++	++	
21	#19	LR	MOL	DRT	++	++	++	0	++	++	+	++	++
6	#22	LL	MOL	DRT	+	++	++	+	++	+++	+	+	++
12	#14	LL	MOL	DRT	+	++	++	+	++	++	+	++	++

Those lesions associated with the mesial roots of the mandibular fourth premolars minimally involved the tissues located on the buccal aspect of the roots very little and appeared to spread almost equally in the other directions.

The lesions surrounding the distal roots of the mandibular fourth premolars were perhaps the most uniform in shape and distribution. There was no apparent predilection for spread in any one direction although the buccal tissues were involved slightly less often than others.

Although the mesial roots of the mandibular first molars were generally located close to the lingual cortical plates, there seemed to be a tendency for the lesions to move in that direction. These lesions also favored a distal spread. In two instances, the buccal tissues appeared unaffected.

The extremely close proximity of the distal roots of the mandibular first molars to the lingual cortical plates apparently had little bearing on the relative distribution of the respective pulpo-periapical lesions. In general, these lesions appeared to spread similarly in all directions.

Table V indicates the most likely relative directions in which the lesions spread with respect to their respective root apices. It is a summary of Table IV. Lesions surrounding the mesial root apices of the fourth premolars favored distal and apical spread. Those surrounding the distal roots of the fourth premolars favored lingual, apical, and mesial distribution. Distal and apical were also the directions in which lesions spread from the apices of the mesial roots of the mandibular first molars. The distal roots of the mandibular first molars followed the same patterns also. These lesions tended to be

TABLE V

DISTRIBUTION OF PULPO-PERIAPICAL LESION TO ROOT APEX

(COMPILED FROM TABLE IV)

Specimen #	Dog #	Side	Tooth	Root	Buccal	Lingual	Apical	Mesial	Distal	Relative Root Apex Position (B-L)
C	Control	LR	PRE	MRT	- - - - No Lesion Present - - - -					Buccal
4	#22	LR	PRE	MRT	+	++	++	++	++	Buccal
16	#14	LR	PRE	MRT	+	++	++	++	+	Buccal
19	#19	LR	PRE	MRT	+	+	++	0	++	Central
9	#22	LL	PRE	MRT	+	+	++	++	++	Buccal*
11	#14	LL	PRE	MRT	+	++	+	++	++	Buccal
					5	8	9	8	9	
D	Control	LL	PRE	DRT	- - - - No Lesion Present - - - -					Central
3	#22	LR	PRE	DRT	+	++	++	++	++	Buccal
17	#14	LR	PRE	DRT	++	++	++	++	++	Central
18	#19	LR	PRE	DRT	++	++	++	+	++	Central
8	#22	LL	PRE	DRT	++	++	++	++	0	Buccal*
10	#14	LL	PRE	DRT	++	++	++	++	++	Buccal
					9	10	10	10	8	
B	Control	LR	MOL	MRT	- - - - No Lesion Present - - - -					Lingual
5	#22	LR	MOL	MRT	0	+	+++	+	+++	Lingual
14	#14	LR	MOL	MRT	++	++	++	++	++	Lingual
20	#19	LR	MOL	MRT	++	+	+	++	++	Lingual*
7	#22	LL	MOL	MRT	0	++	++	+	+++	Central
13	#14	LL	MOL	MRT	+	+	++	+	+++	Lingual
					5	7	10	7	13	
15	#14	LR	MOL	DRT	+	++	++	+++	+	Lingual*
21	#19	LR	MOL	DRT	++	++	++	++	++	Lingual*
6	#22	LL	MOL	DRT	+	++	++	+	++	Lingual*
12	#14	LL	MOL	DRT	+	++	++	++	++	Lingual*
					5	8	8	8	7	

*Indicates Root Apex Very Close or Touching Respective Cortical Plate

directed lingually, apically, and mesially more often than other directions. The relative positions of individual root apices with respect to the cortical plates of bone were reiterated for reader convenience.

As indicated in Table VI, pulpo-periapical lesions differed in size depending upon the technique used to measure them. Lesion size was measured with a millimeter ruler in the case of radiographs. Radiographs were exposed from the buccal *in vivo* and on specimens. They were also taken from the mesial of the specimens. The histologic sections were buccal-lingual and viewed from the mesial. The size of the lesions in these cases was measured in microns by counting the 5 micron (μ) sections taken.

The average size of the lesions measured by the various techniques from mesial to distal indicated the following:

Lesion Size on Specimen Radiograph > Lesion Size on *in vivo* Radiograph > Lesion Size on Histologic Sections

The average lesion size on *in vivo* radiographs was an average of 7.5% larger than the same lesion measured from histologic sections. The average lesion size measured from specimen radiographs (individual block sections of a root, lesions, and surrounding bone) devoid of all soft tissue, were found to be 15.5% larger than the histologic sectioning measurement (Table VI). Therefore, it was concluded that there must be some factor responsible for this unexpected result. It was then decided to determine if image magnification was involved in this discrepancy.

Table VII has shown that a magnification factor existed in this study. Measurement between the same two points on the specimen radiographs and of the histologic sections indicated an 11.2% larger average

measurement by radiographic means. Therefore, it can be stated that a magnification factor partially explained the reversal of outcome expected in this study concerning lesion size.

TABLE VI

RELATIVE SIZE OF LESIONS VIA DIFFERENT MEASUREMENT TECHNIQUES

Specimen #	Dog #	Side	Tooth	Root	Size of Lesion (Radiograph of Specimen)	Size of Lesion (Microtome Count)	Size of Lesion (On Radiograph In Live Dog)
3	#22	LR	PRE	DRT	≃ 4.0 mm	3500 μ	4.0 mm
4	#22	LR	PRE	MRT	≃ 4.0 mm	3200 μ	3.5 mm
5	#22	LR	MOL	MRT	≃ 6.0 mm	4600 μ	5.5 mm
6	#22	LL	MOL	DRT	≃ 5.0 mm	4800 μ	4.5 mm
7	#22	LL	MOL	MRT	≃ 4.5 mm	4400 μ	4.0 mm
8	#22	LL	PRE	DRT	≃ 4.0 mm	4400 μ	3.5 mm
9	#22	LL	PRE	MRT	≃ 4.0 mm	3200 μ	4.5 mm *
10	#14	LL	PRE	DRT	≃ 4.0 mm	3000 μ	5.0 mm
11	#14	LL	PRE	MRT	≃ 4.0 mm	**	4.0 mm
12	#14	LL	MOL	DRT	≃ 7.0 mm	7200 μ	5.0 mm
13	#14	LL	MOL	MRT	≃ 5.5 mm	4000 μ	5.0 mm
14	#14	LR	MOL	MRT	≃ 5.5 mm	4600 μ	5.0 mm
15	#14	LR	MOL	DRT	≃ 4.5 mm	3800 μ	4.5 mm
16	#14	LR	PRE	MRT	≃ 3.5 mm	2800 μ	3.5 mm
17	#14	LR	PRE	DRT	≃ 5.0 mm	4200 μ	4.5 mm
18	#19	LR	PRE	DRT	≃ 3.0 mm	2700 μ	4.0 mm
19	#19	LR	PRE	MRT	≃ 2.5 mm	3200 μ	2.0 mm *
20	#19	LR	MOL	MRT	≃ 5.5 mm	4600 μ	5.5 mm
21	#19	LR	MOL	DRT	≃ 4.0 mm	3400 μ	4.0 mm
B	Mesial Rt. 1st Molar (Normal)						
C	Mesial Rt. 4th Bicuspid (Normal)						
D	Distal Rt. 4th Bicuspid (Normal)						
				Averages:	89.5/19=4.7mm	(3970μ) 71.5/18=3.97mm	81.5/19=4.29mm
					4.7 mm (15.5% larger)	> 3.97 mm <	4.29 mm (7.5% larger)

*Ill defined

**No count possible

TABLE VII

OBSERVED MAGNIFICATION FACTOR UTILIZING

FIXED POINTS* ON THE SPECIMEN FOR MEASUREMENT

SPEC. #	I MILLIMETER MEASUREMENT-- SPECIMEN RADIOGRAPHS	II MICRON MEASUREMENT-- HISTOLOGIC SECTIONS
C	No measurements	
4	4.00	2450
16	3.25	2400
19	4.00	3800
9	3.50	2800
11	5.50	6600
D	No measurements	
3	2.00	1950
17	3.00	1800
18	2.00	1600
8	4.50	3400
10	3.00	2300
B	No measurements	
5	5.00	4200
14	3.50	1400
20	5.50	5000
7	3.00	2400
13	5.50	4600
15	5.00	2400
21	3.75	1400
6	4.00	4600
12	6.00	4800
	76/19=4.00	59.9/19=3.15
Average:	4.00 mm	3.15 mm

Per Cent Magnification Observed: 11.2%

*The Distance Between The Same Two Points Was Measured For Each Experimental Specimen

CHAPTER V

DISCUSSION

The specific purposes of this paper were the following:

1) To describe the normal radiographic and histologic features which existed in the dentoalveolar complex of the dog mandible;
2) To describe the radiographic and histopathologic features which existed in the presence of pulpo-periapical lesions in the dog mandible; and,
3) To compare the radiographic and histologic appearance of pulpo-periapical lesions in the dog mandible.

Concerning the normal appearance of the dog mandible, there was agreement and disagreement with the findings of others.[18,24] Seltzer and Bender[18], and Pauls and Trott[24], described the location of the roots of dog teeth with respect to the cortical plates of bone. Both of these studies agreed that the position of the roots of dog teeth were approximately the same as in the human mandible. They stated that there exists a shift from buccal to lingual in the location of root apices as one progresses distally along the mandible of a dog or a human. This was, in general, found to be the case. However, an important distinction was observed. The first molar root apices of the dog compared in their lingual inclination and position more closely to the second or third molar roots of the human. Clinically this may have been of some significance. A perforation of the lingual cortical plate in this area of

the human mandible could allow the spread of infection into the sublingual and submandibular spaces along fascial planes. This is due to the more anterior and superior attachment of the mylohyoid muscle in this area. Since the first molar was found to be located in a more anterior position in the dog mandible than is its human counterpart, differences in muscle attachments and subsequent differences in the spread of dental infection may exist. Therefore, anatomic location and distribution of structures important to spread of infection would need to be analyzed carefully prior to comparisons with the human condition.

Schalle[35] stated that the roots from mesial of the fourth premolar to distal of the first molar became progressively longer in the dog. This was not found to be the case in this study with respect to the distal root of the first molar. It was found to be shorter in all cases than the mesial root of the first molar (Fig. 19).

Due to the results of earlier studies[40,44,49], it was expected that certain radiographic and/or histologic features might be observed again. A thickened apical periodontal membrane space including substantial vascularity and loose connective tissue was found in normal specimens as was a layer of secondary cementum around their roots (Fig. 5). The possibility that the carnivorous nature of the dog, discerned by the cutting nature of the posterior teeth, may be responsible for these differences from the dental complex of the human omnivore was considered.

Another interesting difference between the human and dog mandible was found to be the thickness of the cortical plates of bone and the relative amounts of medullary bone present. The dog mandible was found to have far less medullary bone and much thicker cortical plates than

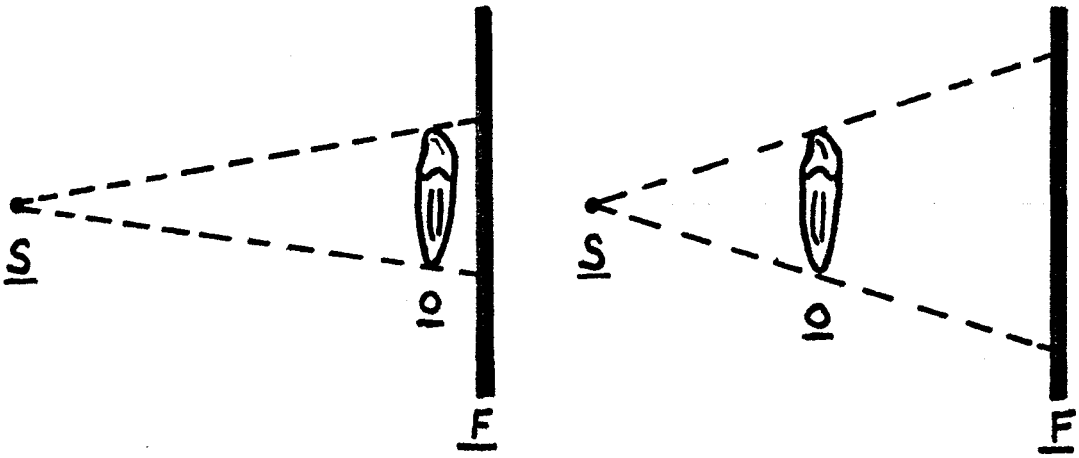
does the human mandible. Perhaps this too relates to dog mandible function. Since bone deposition has been related to functional stress, it is possible that the dog mandible has far greater strength than does the human mandible. This discrepancy was not anticipated prior to the undertaking of this experiment. In all cases (Fig. 20), an intimate relationship existed between the root apices and the mandibular canal of the dog. Due to this fact and along with the observation of a decreased number of marrow spaces observed in the dog mandible as compared to the human, it was assumed that the loose connective tissue surrounding the major structures in the mandibular canal was fatty marrow capable of cellular proliferation.

Due to the many and varied differences in the physical structure of the dog dentoalveolar complex as compared to the human, it was determined that the dog may not be an acceptable model for comparison of normal tissues.

Many similarities were observed when comparing the properties of pulpo-periapical lesions in dogs to those of humans. It was determined that necrosis, cellular infiltrates, bone resorption, root end resorption, disruption of periodontal membrane space, encapsulation, and bone remodelling all occur under the proper circumstances in the dog. According to Torneck and Tulananda[44], bone apposition was a direct response of the periodontal membrane to infection viewed after 83 days. There was no direct evidence in the present study to support or deny this contention. They also described bone apposition around the periphery of a lesion except where cellular infiltrate was intense. This was definitely the case in the present study as well. It seemed as though there was not only a fibrous encapsulation of the most intense portion of each lesion, but also an attempt by bone to accomplish the same thing.

Bone resorption and root end resorption were commonly observed in the histologic sections. The resorption most often appeared ragged and without uniformity in the apical segment of the root. Apparently, osteoclast-like cells in the dog have no specificity. Bone apposition or remodelling, on the other hand, stained lighter with H & E, appeared more cellular than surrounding bone, and consisted of gentle curves indicative of new lamellae construction. For deposition of bone to occur around the lesion brought more suspicion upon the acceptability of the dog as a model to be compared to the human. Others[41,44] have indicated that resorption and deposition patterns of bone were not the same as in the human, nor was progression of a lesion in the dog found to be the same. Those same authors went on to say that the histologic aspects found in the dog and in the human were similar enough to allow endodontic research on the dog. The only conclusion which could be drawn was that periapical pathosis causes an increase in bone deposition in the dog mandible at some distance away from the central necrosis.

The comparison of lesions radiographically and histologically was a very difficult task. The average size of all pulpo-periapical lesions in this study was found to be smaller histologically than radiographically. This is in contrast to other studies.[13,14,15,23] Table VII attempts to partially explain why this occurred in the present study. However, magnification error alone cannot be blamed for these results. Technical errors per se, though many were possible and surely included, as well as subjective error in the application of technology and analysis of results, led to the discrepancies observed. Two of those possible errors are illustrated on the following page:



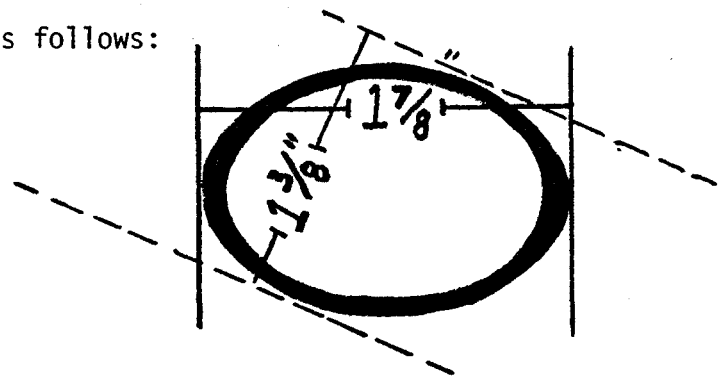
Changes in source to object distance, and/or object to film distance, cause image size discrepancies on the radiograph. These variables must be standardized so that they may remain the same for each quadrant of each and every animal. They must also be accounted for in any measurements made of radiographs taken of block specimens following sacrifice. If not, errors in image size measurement will surely result.

Subjective errors included an inability to determine that point on a histologic section at which resorption of bone ceased being of great enough magnitude to appear radiographically. Neither demineralization nor lack of viability of bone was visible on the dental radiograph. Histologically, this may also have been the case. Even then, Duinkerke and associates[56] showed errors of 21% - 37% when measuring the same radiographically visible lesion with a millimeter ruler. Considering the diffuse nature of lesion borders encountered on radiographs in this study, it was surprising that results this consistent were obtained.

Subjective error in application of technology was perhaps the most difficult aspect of this study to monitor and control. Most importantly, this included "positioning error" as described by Duinkerke

and van de Poel.[55] These authors showed that apparently identical radiographs, taken at slightly different angles, caused the size of lesions on the radiographs to differ. Also, they showed that "positioning error" caused differences in measurement of the histologic lesions and the radiographic lesions. With soft tissue an ever present variable, it was no wonder that small differences in angulation were recorded on apparently identical radiographs. Utilizing far more sophisticated equipment than this study allowed, it would still have been extremely difficult to have duplicated technical factors from one experimental period to another, or even from one animal to another. For example, to ensure that the x-ray beam was perpendicular to the greatest breadth of any given lesions would have been nearly impossible. For these reasons, there existed an inherent subjective error which corresponds to the clinical application of radiology in dentistry. Although actual sizes were assigned to all radiographically visible lesions in this study, it has become apparent that pulpo-periapical lesion size is a difficult entity to measure on a dental radiograph.[55]

Purely technical factors also caused error in the determination of true pulpo-periapical lesion size. If the plane of sections taken from the block specimens were not perpendicular to the greatest breadth of the lesion, then unless the lesion was perfectly circular, the microtome could not reveal its true size in microns. This concept can be illustrated as follows:



When measuring an oval, there is only one way to obtain its greatest diameter and that is by measuring the distance between perpendicular lines drawn at the greatest diameter of the oval. Measurement between any two other points would yield a lesser diameter. The same concept surely applies to the non-uniform shape of a pulpo-periapical lesion.

Only the distal root of the fourth premolar exhibited anything which resembled a circular lesion. This was the only root studied histologically which showed no aversion to any particular direction in so far as lesion spread was concerned. In contrast, Smith[33] stated that pulpo-periapical lesions, in general, were found to be circular due to the fact that nothing exists to prevent spread in any and all directions. The present study demonstrated that this opinion does not hold true for rapidly expanding lesions.

From the foregoing discussion and during the latter stages of the experiment, it became apparent that only an adherence to strict paralleling techniques had any chance at minimizing the built-in variables. Therefore, after having infected and radiographed the maxillary teeth of the dog, it became apparent over the course of the experiment that they would not be of value in the present study. The very flat hard palate of the dog made even the bisecting angle technique very difficult to perform. The resultant images were foreshortened considerably. If experimentation in this direction is attempted, more dogs and only the mandibular teeth should be utilized. Only in this way can a reasonable sample size be obtained.

In the final analysis, it was safe to say that the radiographic appearance of a pulpo-periapical lesion had little relation to its respective histologic appearance. In other words, our clinical ability

to view a lesion on the radiograph is a relative diagnostic tool and cannot be relied upon as the only means with which to determine the absence or presence of a lesion, let alone its actual size.

Some mention must be made concerning an initial premise of this study which had to be eliminated. This relates to the previously described difficulty encountered in classifying what consituted or did not constitute a lesion. Bender and Seltzer[17,18], Garber[21], and Ramadan and Mitchell[22] stated that junctional trabeculae must be destroyed or more serious damage to the inner or outer cortical plate must occur before a lesion may appear radiographically. Regan and Mitchell[20] defined junctional trabeculae as "ridges left after the central marrow is gone." It may be possible to detect histologically when this point is reached in a human mandible study, but it was the opinion of the investigator of this study that junctional trabeculae cannot be demonstrated in a dog mandible. There existed very little marrow between the large and dense bone spicules which could be called medullary bone. Never were any "ridges left"[20] observed which could have been called junctional trabeculae. Perhaps a more quantitative determinant of bone loss (such as demineralization) occurred in order that the dog lesions appeared radiographically.

The inital premise eliminated in the early stages of this study was an attempt to determine at what stage a pulpo-periapical lesion first became visible on the dental radiograph. Technique factors again were a problem, as was the feasibility of establishing the criteria for determining the presence of a lesion. Without unlimited funds for the purchase of equipment to allow the use of variable kilivoltage peak (KVP) and milliamperage seconds (MaS) throughout a wide range of

parameters to be used, neither initial appearance nor criteria could be established. Nevertheless, an early attempt was made to carry out this portion of the experiment with the S. S. White Flexomatic 90 x-ray machine available for use. This machine was purported to be a variable KVP and MaS machine in clinical usage. The advantage of this type of variable machine should be that low KVP, high contrast films could be taken when necessary, using a longer exposure time to accommodate the greater number of long wavelength x-rays emitted. The longer wavelength x-rays have poor penetrating ability and are apparently absorbed in high doses by the skin upon entrance. Higher numbers of x-rays must be produced in order to expose the intraoral film. The opposite was also expected. Using a higher KVP, a lower contrast image should have been available, which had a longer grey scale and therefore subtle density changes chould be more easily observed. The higher KVP technique should have emitted more hard x-rays of short wavelength which are more penetrating in nature and believed to be absorbed in greater numbers in deeper structures. It is possible that this combination could affect more sensitive tissues such as bone marrow, lymphoid, or glandular tissues, in a detrimental manner.

The major problem in this experiment was that this machine had inherent filtration equivalent to 2.5 mm of Aluminum. This amount of filtration corresponded to the necessity of using a high KVP technique to obtain acceptable film images. Therefore, unreasonable and clinically unacceptable MaS were required to obtain proper exposure on a film using other than 90 KVP. Perhaps a low KVP technique with resultant high contrast would detect a pulpo-periapical lesion before a high KVP,

low contrast technique. Further research in this direction should be undertaken in order to determine the validity of this premise.

Another problem which was encountered early in the detection study was even more unpredictable in nature. Apparently, due to their virulence, pulpo-periapical lesions began to appear radiographically one week after infection with Streptococcus faecalis. According to other investigators[42,49,51], it required between three and eight weeks to detect pulpo-periapical lesions radiographically. Apparently, it would have been prudent to run a separate pilot study on one dog to determine the proper dilution of this strain of microorganism in order to induce a slowly developing lesion *in vivo*. This lesion could then have been radiographed in serial fashion and variable KVP and MaS could have been used to determine the first indications of periapical radiolucency.

There were several procedure changes which chould have been made to simplify or improve the experimental design.

One dog expired following intravenous injection (IV) of an apparent overdose of anesthetic. Apparently, additional anesthesia, without fear of overdose can be obtained by IV injection on the ventral surface of the tongue of the dog. This method may have been an easier and safer method with which to maintain adequate operative anesthesia. When the Beagle began to awaken from general anesthetic, he entered a stage of excitement or irritation. At this time a painful stimulus (needle puncture) provoked a great deal of reflex movement making further attempts at injection more difficult. At the same time, the animal emitted a noise which can only be described as screaming, which made the experience all the more disturbing and embarrassing for the experimenter.

Another factor related to maintenance of anesthesia should be discussed. Apparently it has been common practice to inject a volume (approximately 2cc) of atropine sulfate subcutaneously following IV induction of barbiturate anesthesia. Atropine sulfate is a cholinergic blocking agent. Along with inhibition of salivary flow, facilitating intra-oral operative procedures, in small doses it stimulates the respiratory mechanism and nullifies bradycardia. This added injection might have prevented the loss of one experimental animal at a critical time in the study.

Some of the histologic sections in this experiment were found to lack detail. Bone and teeth require substantial time to decalcify in 5% formic acid solution. Some experimenters have waited over three months for decalcification of specimens in pathology laboratories. Decalcification of specimens occurred in 5-9 days with the solution used, depending on the individual block sample size. Cellular detail was lost in the specimens so that only general observations could be made. It would be prudent for any researcher to take the extra time necessary to try and obtain good detail so long as histologic investigation is required as part of the experimental procedures.

Staining methods should also be varied in an experiment of this type. According to Hill[40], dog periapical tissues are easier to infect than human periapical tissues due to the multiple foramina present in the tooth of a dog. If this is true, bacteria should pass freely into the periapical tissues of the dog. It might have been of some value to gram stain several histologic sections for the presence of bacteria. Also, some sections should have been stained specifically for the presence

of inflammatory cells if possible. These staining techniques would lend credibility to any experimental attempts in this direction.

Perhaps the major conclusion of this study has been previously stated. Coolidge[38] and Orban[39] have both expressed the opinion that it is extremely difficult to relate one experimental model to another. From the foregoing results and discussion, it is believed apparent that the dog may not be an acceptable animal model for research directed toward the study of pulpo-periapical lesions. At any rate, human comparisons would not seem valid.

CHAPTER VI

SUMMARY AND CONCLUSIONS

The Beagle dog was found to have some advantages and many disadvantages with respect to research directed toward the study of pulpo-periapical lesions.

The morphology and size of the fourth premolar and first molar teeth were similar to the human, so that routine armamentarium was adaptable to the animal. In general, the dogs were able to withstand experimental procedures well, and the availability, cost, and care of the animal were reasonable. With regard to lesions, it seems reasonable to state that dog periapical tissues are easy to infect with pathogenic microorganisms such as Streptococcus faecalis.

However, the dog was found to exhibit major disadvantages as an acceptable experimental model. The bony architecture of the dog mandible was found to be very different from the human counterpart. The cortical plates were much thicker in the dog and very dense. Very little medullary bone was evidenced, as were very few marrow spaces seen. This is in contrast to the human mandible. The root apex of the first molar of the dog was found to exhibit a very wide periodontal membrane space both radiographically and histologically. Therefore, early detection of a pulpo-periapical lesion on the radiograph was, at best, a relative exercise. Also, the roots of the posterior teeth of the Beagle were found to have an intimate relationship with the mandibular canal. Histologic evaluation of the associated lesions indicated that the

mandibular canal was commonly invaded and this circumstance is unlikely in the human mandible. The maxillary teeth of the dog were shown to be useless for endodontic research due to the flat palate which is present in the animal.

This study also confirmed that the root apices of the posterior teeth of the Beagle are not constant in their positions. From anterior to posterior, the root apices of the fourth premolar and first molar are found to shift in location from a more buccal to a more lingual position with respect to the cortical plates of bone. In general, this is in keeping with that which is found in the human mandible.

Perhaps more importantly, this study seemed to indicate a disparagy between the accepted histologic and radiographic size of a pulpo-periapical lesion. In spite of the fact that measurement techniques and angles may not have been totally accurate, an inverse proportion seemed to exist. The radiographic size of the lesion was generally larger than the histologic analysis showed it to be.

Therefore, it seems reasonable to conclude that a pulpo-periapical lesion may be radiographically visible prior to extensive damage to the cortical plates of the mandible of the dog.

The pulpo-periapical lesion is a far more difficult and complicated entity to study than might be expected at first glance. Many variables were inherent in the technique factors utilized, and each must be standardized in order to validate further research in this direction.

REFERENCES

1. Glenner, R. A.: 80 Years of Dental Radiography; J. Amer. Dent. Assoc. 90:549-563, 1975.

2. Coolidge, E. D.: The Diagnosis and Treatment of Conditions Resulting from Diseased Dental Pulps; J. Nat. Dent. Assoc., pp. 337-343, Dec. 1918.

3. Nyman, J. E.: Editorial Reply to Coolidge - Diseased Dental Pulps; J. Nat. Dent. Assoc., p. 343, 1918.

4. Miller, S. C., and Peltzer, R. H.: An Original Classification of Alveolar Types in Periodontal Disease and Its Prognostic Value: Corroboration by Plasma Phosphatase Determination; J. Amer. Dent. Assoc. 26:565-574, 1939.

5. Barr, J. H.: The Scope and Limitations of Roentgenography as a Diagnostic Procedure; Dent. Clin. N. Amer., pp. 379-389, July, 1961.

6. Bohannan, H. M., and Saxe, S. R.: Periodontal Roentgenographic Interpretation; J. Tenn. Dent. Assoc. 45:406-416, 1965.

7. Bradley, R. E.: Roentgenographic Interpretation of Periodontal Pocket Depth; Chron. Omaha Dent. Soc. 23:20-22, 1960.

8. Prichard, J.: The Role of the Roentgenogram in the Diagnosis and Prognosis of Periodontal Disease; Oral Surg. 14:182-196, 1961.

9. Ball, E. L.: Radiography and Photography in Periodontics; Dent. Radiogr. Photogr. 22:61-66, 76-77, 1949.

10. Frolich, E.: Utilization and Evaluation of Roentgenograms in Periodontal Disease; Dent. Abst. 1:592-594, 1956.

11. Selecky, J. A.: Use of Radiographs in Periodontics; Georgetown Dent. J. 19:16-18, 1953.

12. Goldman, H. M., and Cohen, D. W.: The Infrabony Pocket: Classification and Treatment; J. Periodont. 29:272-291, 1958.

13. Ardran, G. M.: Bone Destruction Not Demonstrable by Radiography; Oral Surg. 24:107-109, 1951.

14. Shackman, R., and Harrison, C. V.: Occult Bone Metastasis; Brit. J. Surg. 35:385, 1948.

15. Goldman, H. M., Millsap, J. S., and Brenman, H. S.: Origin of Registration of the Architectural Pattern, the Lamina Dura, and the Alveolar Crest in the Dental Radiograph; Oral Surg. 10:749-758, 1957.

16. Sicher, H.: Some Principles of Bone Pathology; J. Oral Surg. 7:104, 1949.

17. Bender, I. B., and Seltzer, S.: Roentgenographic and Direct Observation of Experimental Lesions on Bone: I; J. Amer. Dent. Assoc. 62:152-160, 1961.

18. _____: Roentgenographic and Direct Observation of Experimental Lesions on Bone: II; J. Amer. Dent. Assoc. 62:708-716, 1961.

19. Schwartz, S. F., and Foster, J. K.: Roentgenographic Interpretation of Experimentally Produced Bony Lesions, Part I; Oral Surg. 32:606-612, 1971.

20. Regan, J. E., and Mitchell, D. F.: Evaluation of Periapical Radiolucencies Found in Cadavers; J. Amer. Dent. Assoc. 66:529-533, 1963.

21. Garber, F. N.: Roentgenolucent Periapical Areas; Oral Surg. 17: 460-466, 1964.

22. Ramadan, A. E., and Mitchell, D. F.: A Roentgenographic Study of Experimental Bone Destruction; Oral Surg. 15:934-943, 1962.

23. Wengraf, A.: Radiologically Occult Bone Cavities, Br. Dent. J. 117:532-536, 1964.

24. Pauls, V., and Trott, J. R.: A Radiological Study of Experimentally Produced Lesions in Bone; Dent. Pract. 16:254-258, 1966.

25. Grant, D., Stern, I. B., and Everett, F. G.: Orbans Periodontics; St. Louis, 1963, The C. V. Mosby Company, pp. 391, 397-398.

26. Elfenbaum, A.: Alveolar Lamina Dura; Dent. Radiogr. Photogr. 31: 21-29, 1958.

27. Manson, J. D.: The Lamina Dura; Oral Surg. 16:432-438, 1963.

28. Van Der Linden, L. W. J., and Van Aken, J.: The Periodontal Ligament in the Roentgenogram; J. Periodont. 41:243-248, 1970.

29. Rees, T. D., Biggs, N. L., and Collings, C. K.: Radiographic Interpretation of Periodontal Osseous Lesions; Oral Surg. 32: 141-153, 1971.

30. Phillips, J. D., and Shawkat, A. H.: A Study of the Radiographic Appearance of Osseous Defects on Panoramic and Conventional Films; Oral Surg. 36:745-749, 1973.

31. Volchansky, A., and Cleaton-Jones, P.: Bony Defects in Dried Bantu Mandibles; Oral Surg. 45:647-653, 1978.

32. Shoha, R. R., Dowson, J., and Richards, A. G.: Radiographic Interpretation of Experimentally Produced Bony Lesions; Oral Surg. 38:294-303, 1974.

33. Smith, N. J. D.: 6. - The Principles of Radiographic Interpretation; Br. Dent. J. 135:117-121, 1973.

34. LeQuire, A. K., Cunningham, C. J., and Pelley, G. B., Jr.: Radiographic Interpretation of Experimentally Produced Osseous Lesions of the Human Mandible; J. Endo. 3(7):274-6, July 1977.

35. Tagger, M.: Endodontics: A Review of the Past and Its Present Status; Alpha Omegan, 60:107-118, 1967.

36. Scoralle, D. L.: An *in vivo* Study of the Penetrability of Endodontic Restorations; Master's Thesis, Loyola University, 1972.

37. Grove, C. J.: Why Formaldehyde Preparations are Contraindicated in Septic Root Canals with a Practical Scientific Method for Treatment of Putrescent Pulps; Dent. Rev., 27:996-1005, 1913.

38. _____: Some Important Causes of Periapical Infection; Dent. Cosmos 58:1333-1352, 1916.

39. Coolidge, E. D.: Reaction of Dog Tissue to Drugs Used in Root Canal Treatment; J. Amer. Dent. Assoc. 19:747-759, 1932.

40. Orban, B.: The Action of Paraformaldehyde on the Dental Pulp; J. Dent. Res. 13:215-216, 1933.

41. Hill, T. J.: Experimental Dental Granulomas in Dogs; J. Amer. Dent. Assoc. 19:1389-1398, 1932.

42. Dixon, C. M., and Rickert, U. G.: Histologic Verification of Results of Root Canal Therapy in Experimental Animals; J. Amer. Dent. Assoc. 25:781-803, 1938.

43. Barker, B. C. W., and Lockett, B. C.: Utilization of the Mandibular Premolars of the Dog for Endodontic Research; Aust. Dent. J. 16:280-286, 1971.

44. Lawson, D. D., Nixon, G. S., Noble, H. W., and Weipers, W. L.: Dental Anatomy and Histology of the Dog; Res. Vet. Sci. 1:201-204, 1960.

45. Torneck, C. D., and Tulananda, N.: Reaction of Alveolar Bone and Cementum to Experimental Abcess Formation in the Dog; Oral Surg. 28:404-416, 1969.

46. Grossman, L. I.: Minimum Number of Microorganisms Needed to Initiate Growth in Culture Media; J. Dent. Res. 45:81-85, 1966.

47. Palmer, G. R., Lazzarotto, J. R., and Weine, F. S.: Paper Point Transfer of Minimal Numbers of Microorganisms from a Prepared Canal *in vitro*; Oral Surg. 42:824-829, 1976.

48. Dubos, R.: Bacteriostatic Action of Certain Components of Commercial Peptones as Affected by Conditions of Oxidation and Reduction; J. Exp. Med. 52:331-345, 1930.

49. Torneck, C. D.: The Role of Microorganisms in Endodontic Disease; Alpha Omegan, pp. 180-186, 1969.

50. Garcia, D. A., Jansons, D., and Kapur, K. K.: Bone-Imaging and Semiconductor Probe Measurements of Technetium - 99m - Polyphosphate in the Detection of Periapical Pathology in the Dog; Arch. Oral Biol. 21:167-174, 1976.

51. Winkler, K. C., and van Amerongen, J.: Bacteriologic Results from 4,000 Root Canal Cultures; Oral Surg. 12:857-875, 1959.

52. Kasle, M. J., and Klein, A. I.: Television Radiographic Evaluation of Periapical Osseous Radiolucencies; Oral Surg. 41:789-796, 1976.

53. Duinkerke, A. S. H., van de Poel, A. C. M., van der Linden, F. P. G. M., Doesburg, W. H., and Lemmens, W. A. J. G.: Evaluation of a Technique for Standardized Periapical Radiographs; Oral Surg. 44:646-651, 1977.

54. Ramy, C. T., and Segreto, V. A.: Apicoectomy and Root Canal Therapy for Exposed Pulp Canal in the Dog; J. Amer. Vet. Med. Assoc. 150:977-983, 1967.

55. McCormick, J. E.: A Study of Factors Affecting Healing of Developing Periapical Lesions in Immature Teeth of Dogs; Master's Thesis, Loyola University, 1979.

56. Duinkerke, A. S. H., and van de Poel, A. C. M.: An Analysis of Apparently Identical Dental Radiographs; Oral Surg. 38:962-967, 1974.

57. Duinkerke, A. S. H., van de Poel, A. C. M., De Boo, Th., and Doesburg, W. H.: Variations in the Interpretation of Periapical Radiolucencies; Oral Surg. 40:414-421, 1973.

58. Sommer, R. F., Ostrander, F. D., and Crowley, M. C.: Clinical Endodontics, Ed. 3, Philadelphia, 1966, W. B. Saunders Co., p. 411.

APPENDIX

Figure 1a - Cone and positioning device in position to radiograph mandibular quadrant (view from the front).

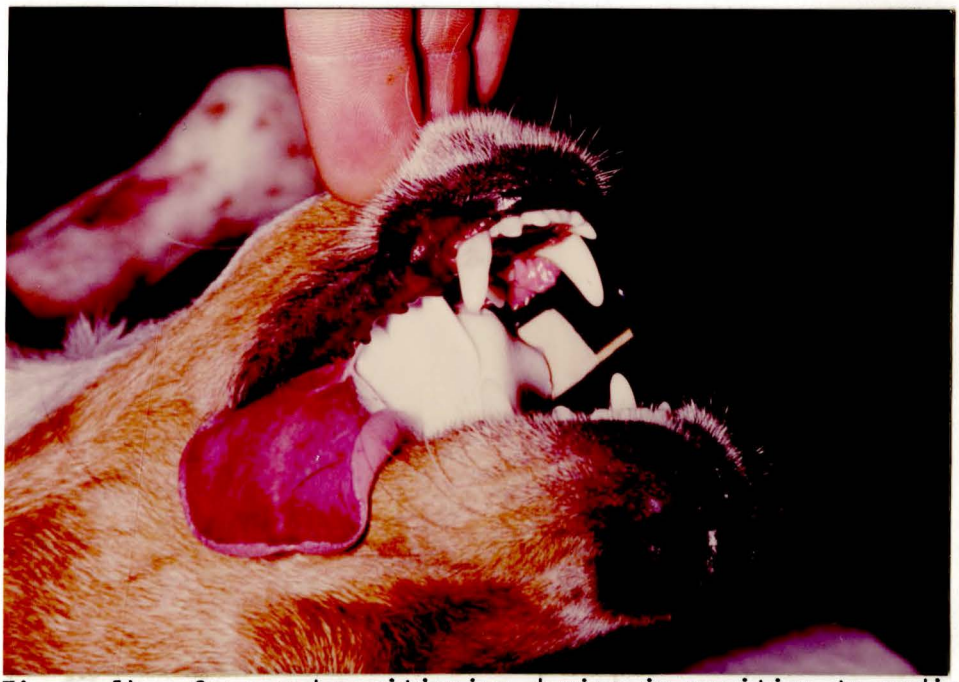


Figure 1b - Cone and positioning device in position to radiograph mandibular quadrant (view from the side).

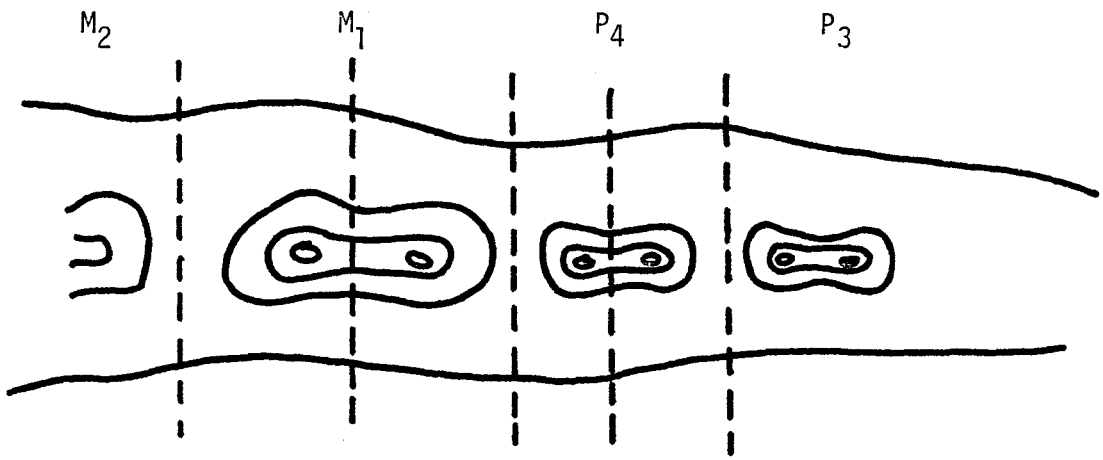


Figure 2 - Plane of buccal-lingual sections which separated each pulpo-periapical lesion into an individual specimen.

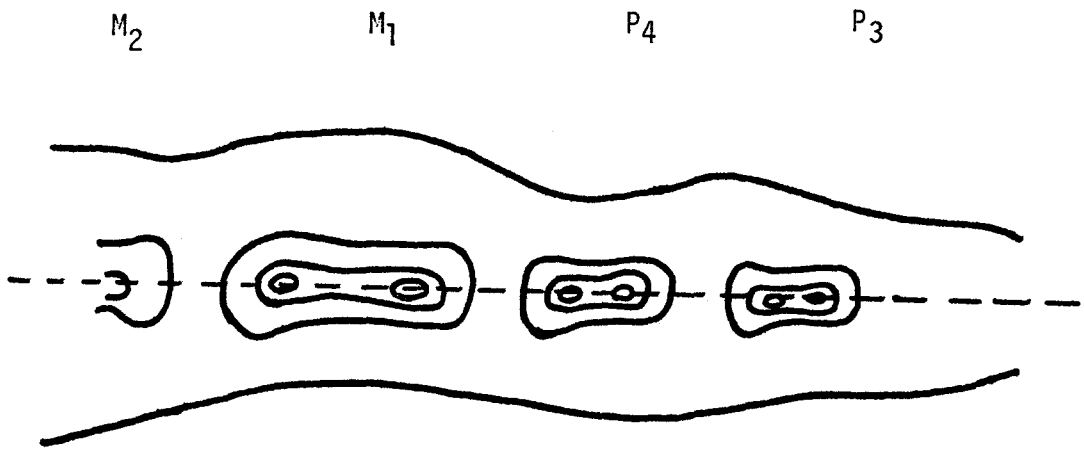


Figure 3 - Plane of mesial-distal section which separated one quadrant block section into two halves through the pulp canals.

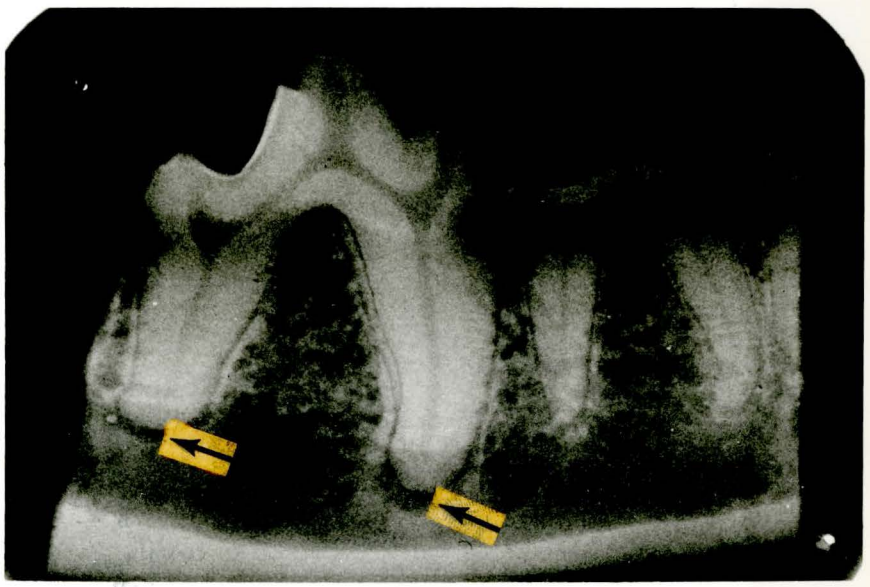


Figure 4 - Radiograph showing the wide PDM spaces which are normal at the root apices of the Beagle 1st molar (arrows).

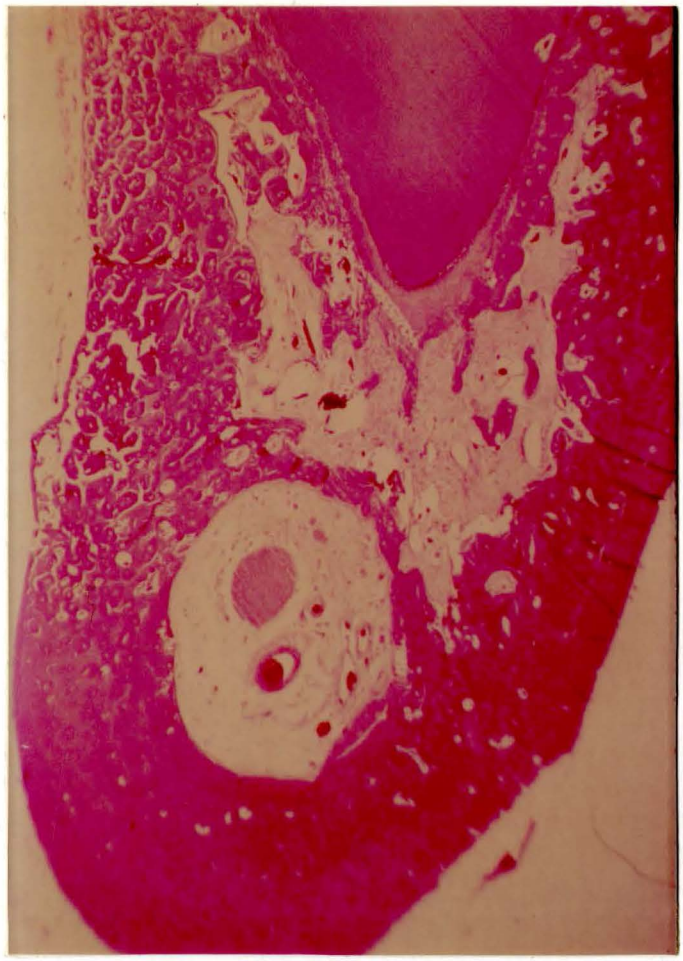


Figure 5 - Photomicrograph showing the wide PDM spaces which are normal at the root apices of the Beagle lst molar. (H & E Stain, orig. mag. x 25)

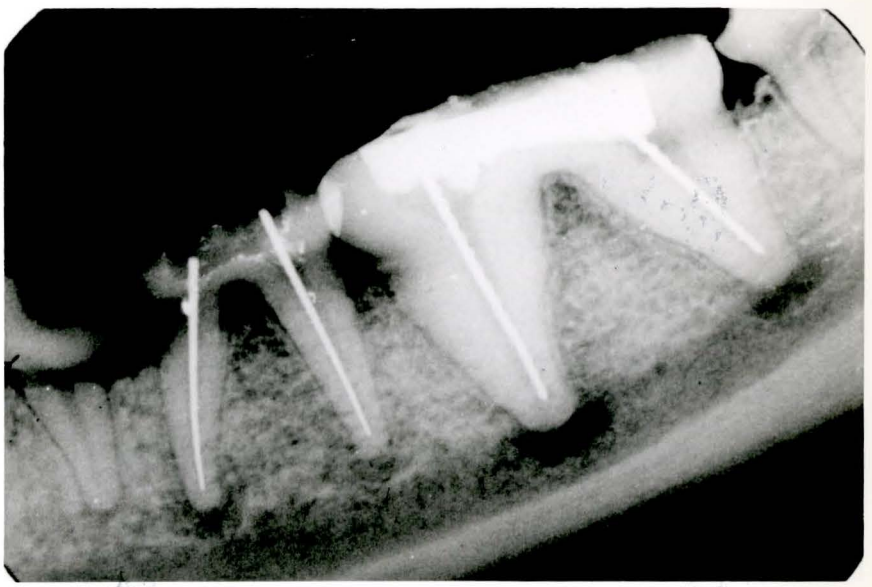


Figure 6 - Radiograph (*in vivo*) indicating the abrupt ending of the main root canals. The files placed in each canal hit a "dead stop" at this location in all cases.

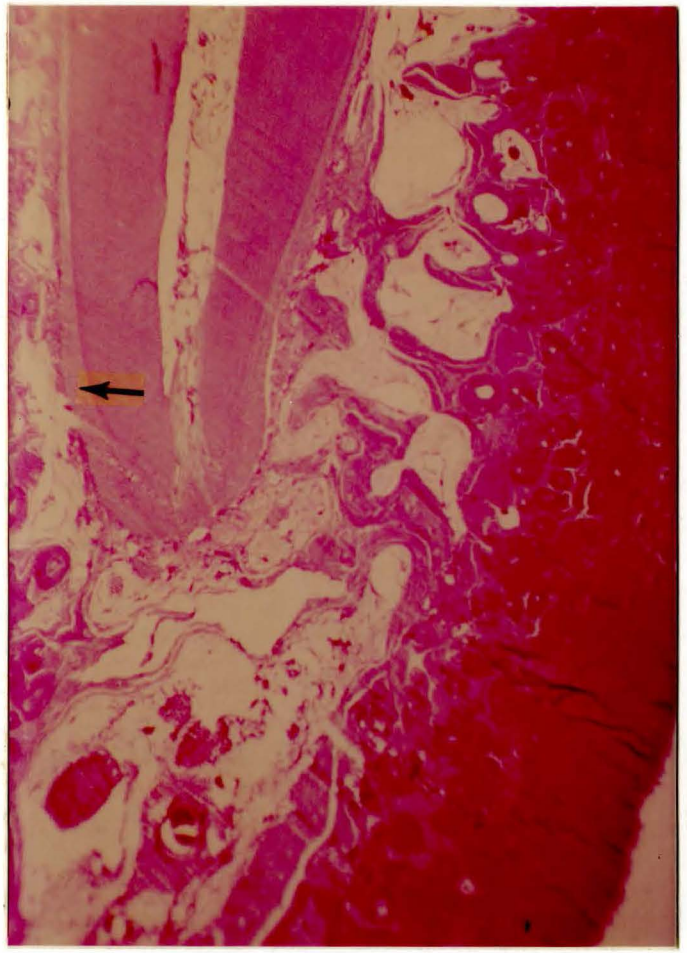


Figure 7 - Photomicrograph showing the apical "Delta-Like" configuration as the canal terminally branched into several smaller canals. Also, a layer of secondary cementum (arrow) was present around the apical 1/3 of all roots examined. (H & E Stain, orig. mag. x 25)

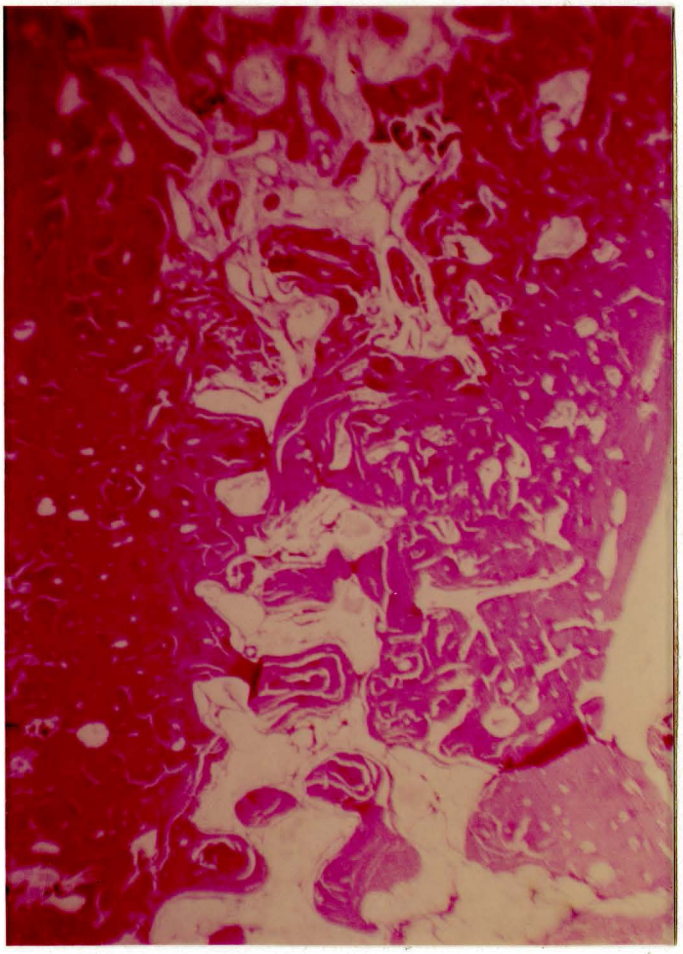


Figure 8 - Photomicrograph showing apparently normal interradicular bone. The cortical plates are extremely thick and dense. A small amount of medullary bone is present and few marrow spaces exist. (H & E Stain, orig. mag. x 25)

Figure 9 - Photomicrograph showing limited component of bone separating mandibular canal contents from adjacent structures above. (H & E Stain, orig. mag. x 25)

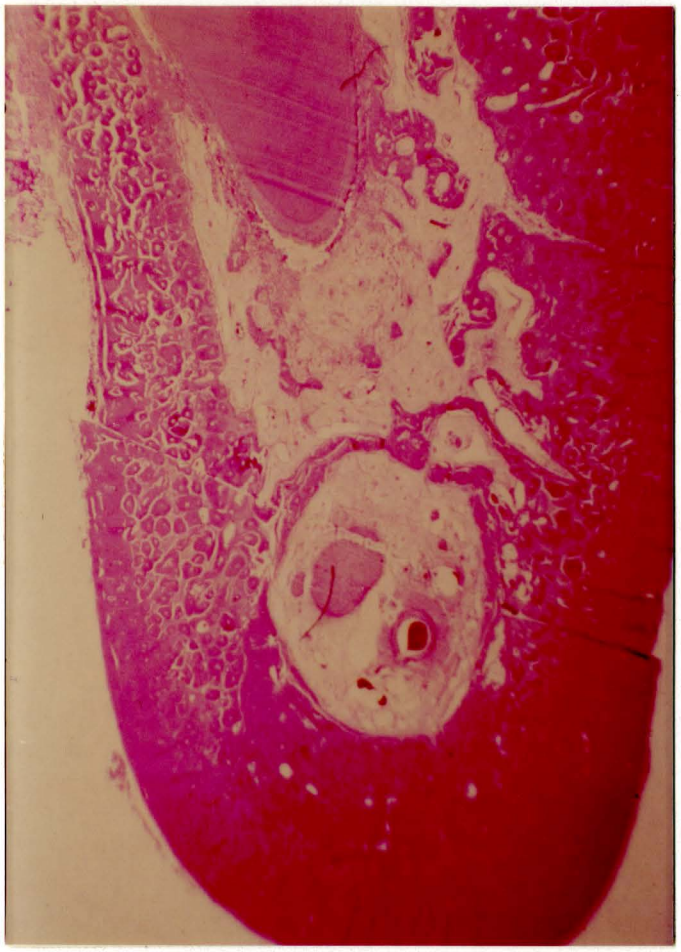


Figure 10 - Photomicrograph showing a thin component of bone separating the mandibular canal from the adjacent lesion and root apex. (H & E Stain, orig. mag. x 25)

Figure 11 - Photomicrograph showing disruption of and communication through the bony demarcation normally separating the canal contents from the adjacent structures. (H & E Stain, orig. mag. x 25)

Figure 12 - Photomicrograph showing nearly complete absence of the bony demarcation between canal contents and existing pulpo-periapical lesion. (H & E Stain, orig. mag. x 25)

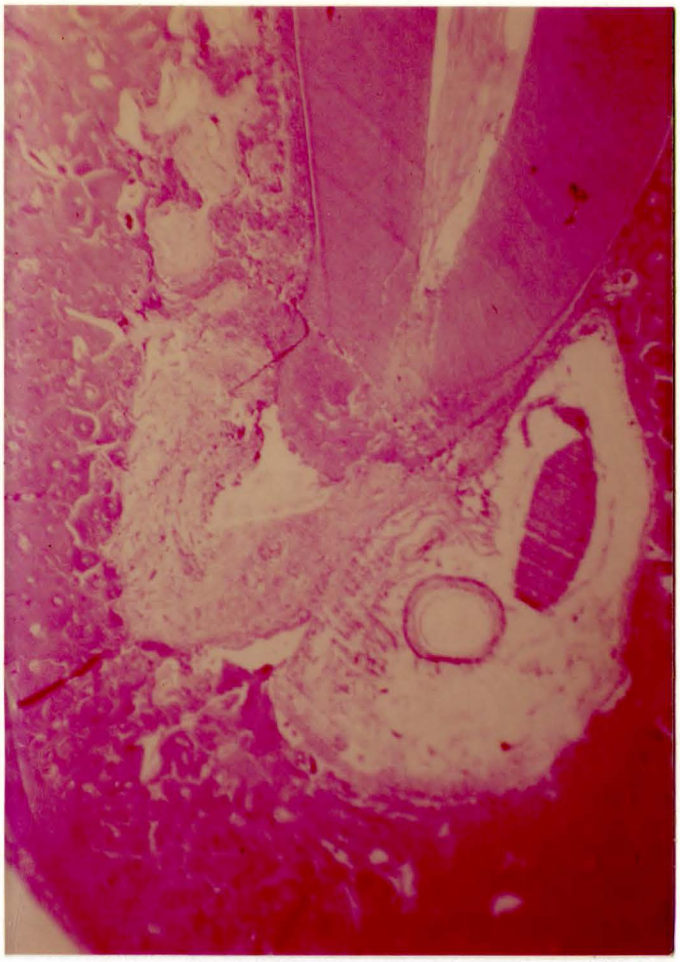


Figure 13 - Photomicrograph showing root end resorption and bone resorption associated with an existing pulpo-periapical lesion. (H & E Stain, orig. mag. x 25)

Figure 14 - Photomicrograph showing bone apposition at the base of the mandibular canal (arrow) in response to an existing pulpo-periapical lesion. (H & E Stain, orig. mag. x 25)

Figure 15

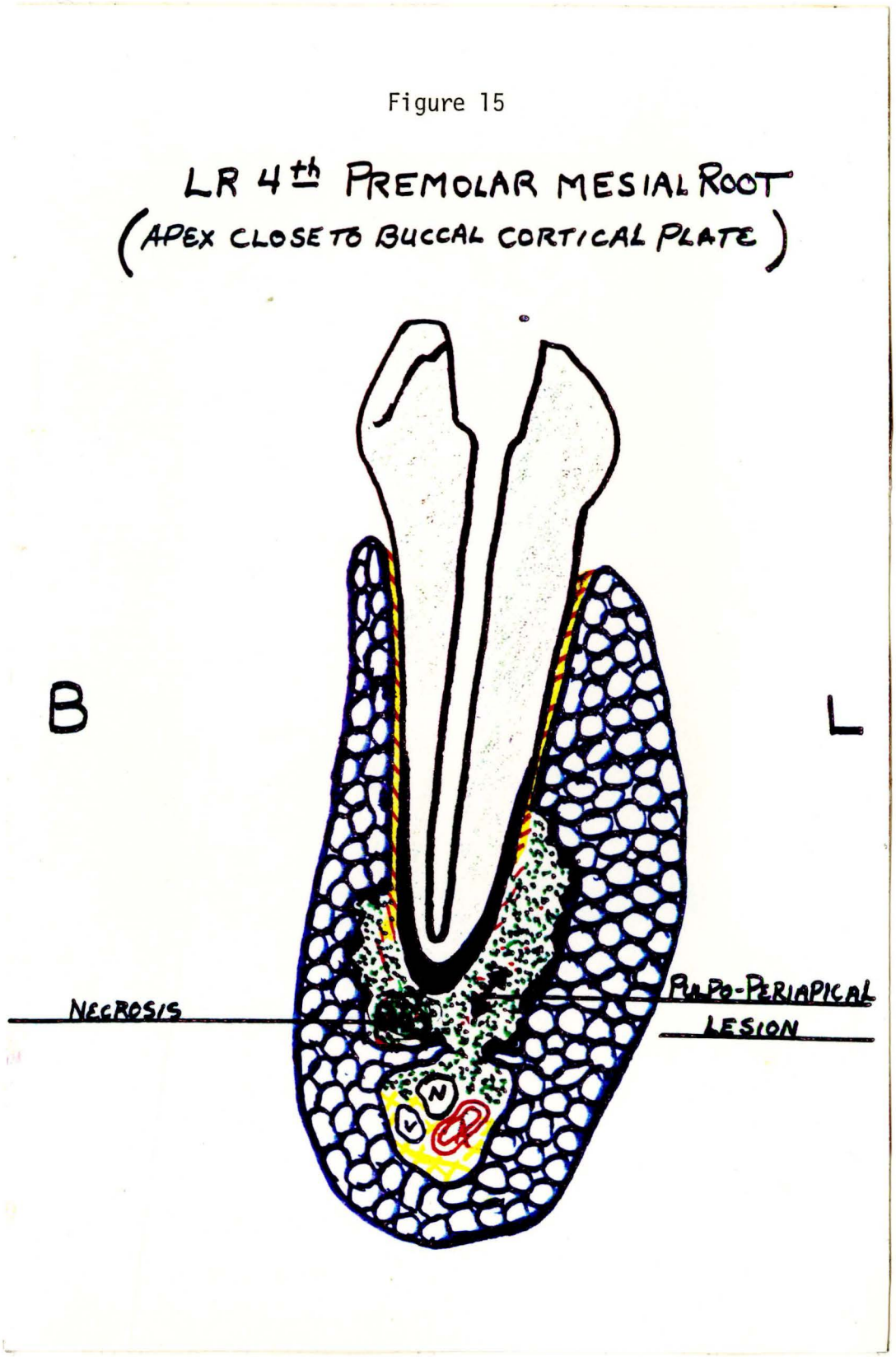

LR 4th PREMOLAR MESIAL ROOT
(APEX CLOSE TO BUCCAL CORTICAL PLATE)
B
L
NECROSIS
PULPO-PERIAPICAL
LESION
N
V

Figure 16

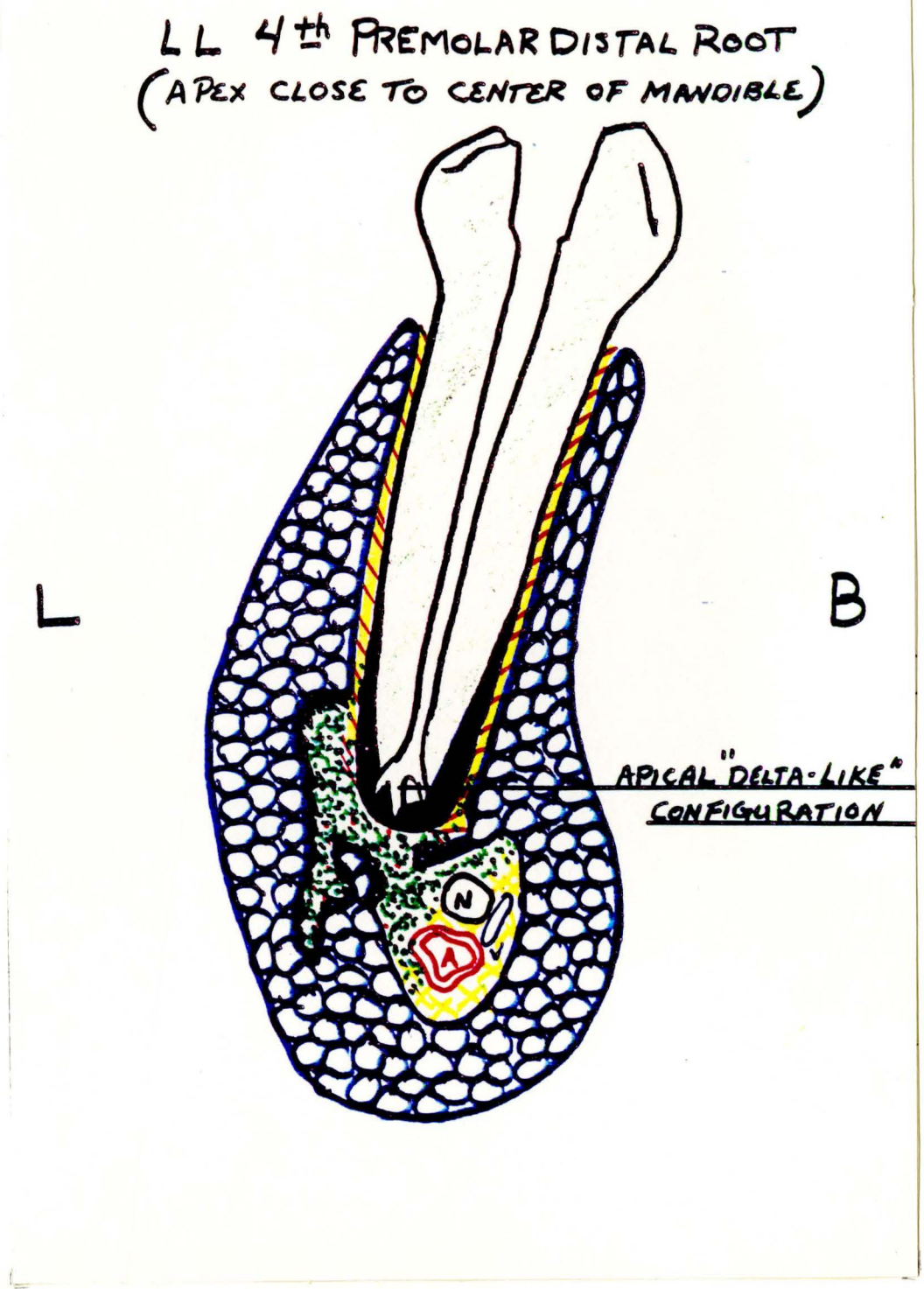

LL 4th PREMOLAR DISTAL ROOT
(APEX CLOSE TO CENTER OF MANDIBLE)
L
B
"APICAL "DELTA-LIKE"
CONFIGURATION
N
A

Figure 17

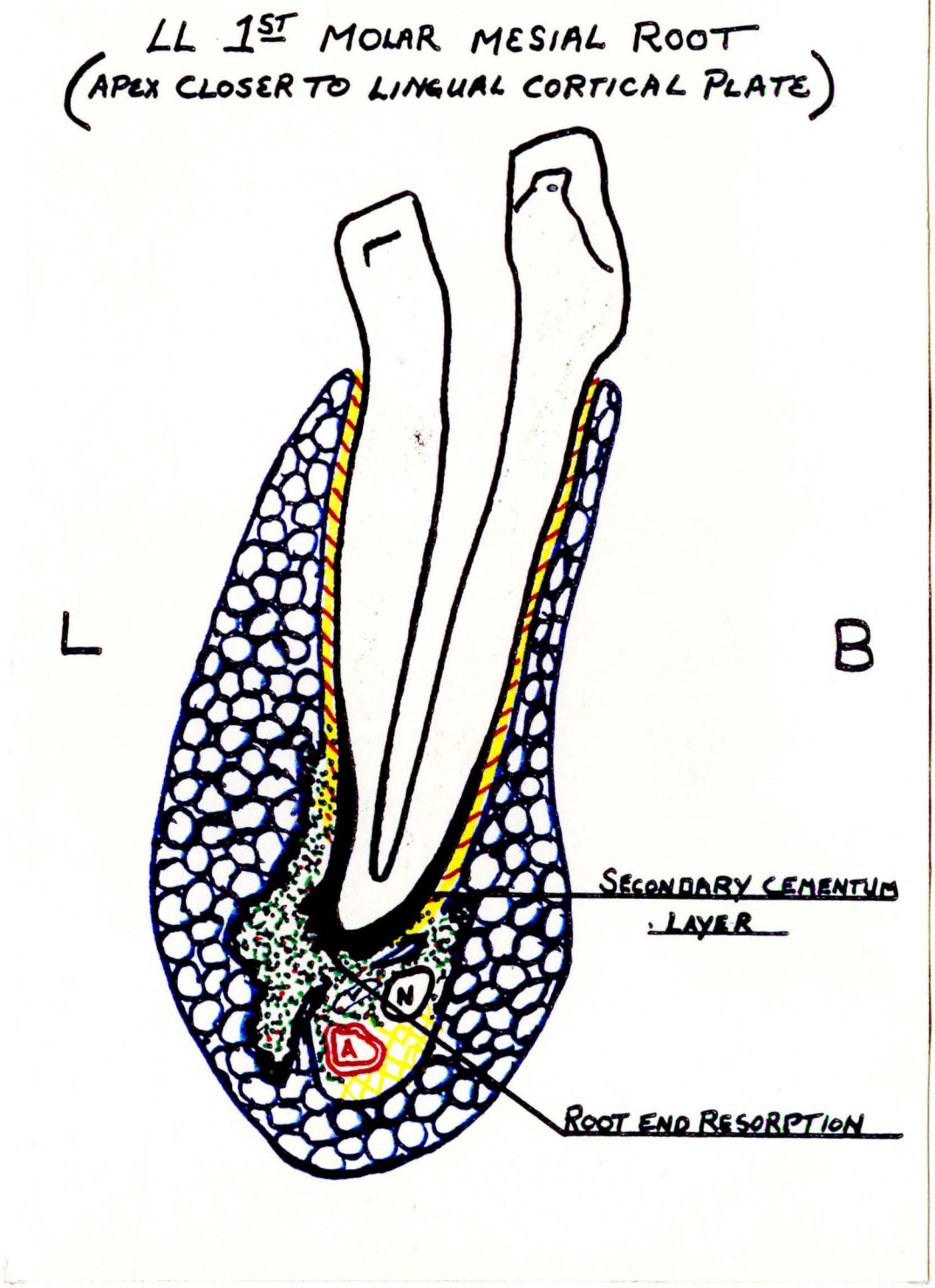

LL 1ST MOLAR MESIAL ROOT
(APEX CLOSER TO LINGUAL CORTICAL PLATE)
L
B
SECONDARY CEMENTUM LAYER
N
A
ROOT END RESORPTION

Figure 18

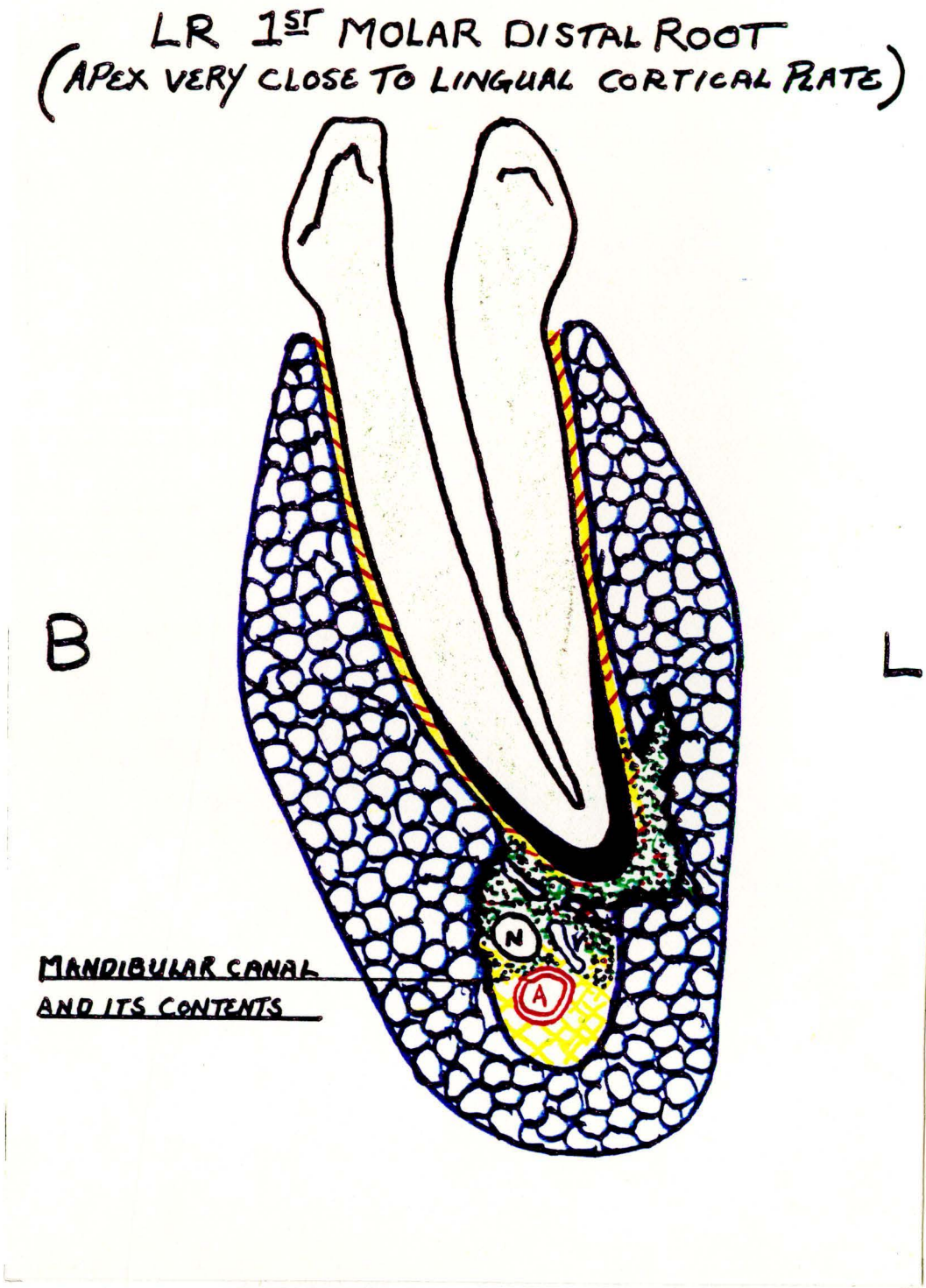

LR 1ST MOLAR DISTAL ROOT
(APEX VERY CLOSE TO LINGUAL CORTICAL PLATE)
B
L
N
A
MANDIBULAR CANAL
AND ITS CONTENTS

Figure 19 - Radiograph showing the distal root of the first molar to be shorter than the mesial root.

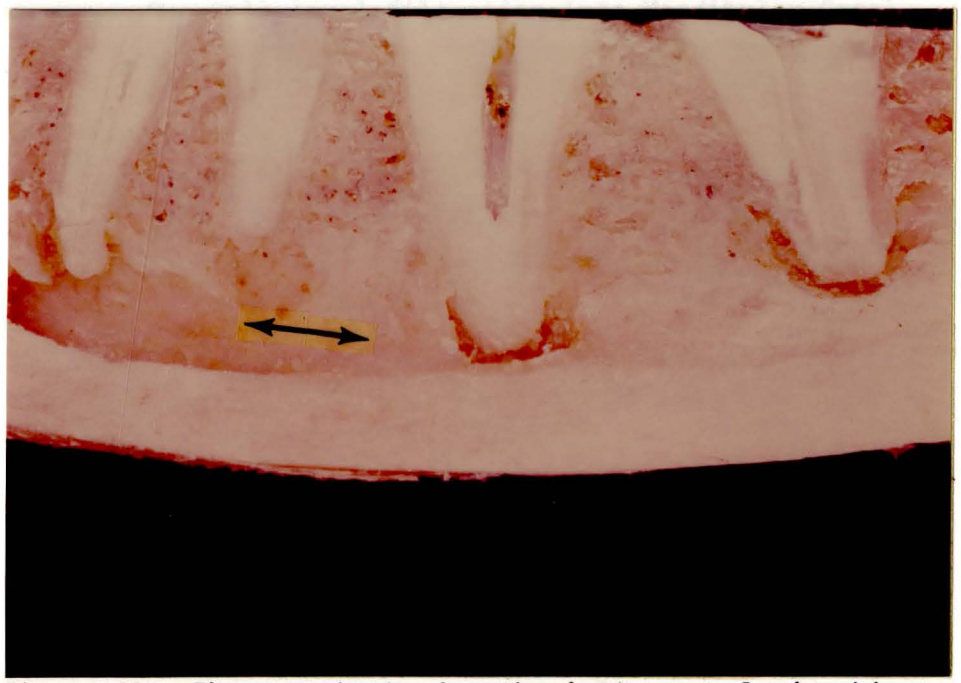


Figure 20 - Photograph showing the intimate relationship between the root apices (as well as associated pulpo-periapical lesions) and the mandibular canal (arrow). (ASA 64, F 16; mag. 1:1)

LEGEND

B - Buccal Cortical Plate Disruption

L - Lingual Cortical Plate Disruption

T - Medullary Bone Trabeculae Disrupted

I - Cellular Infiltration Into Marrow Spaces

C - Cellular Infiltration Into Mandibular Canal

N - Architecture Appeared Normal

TABLE VIII

PERIAPICAL BONE INVOLVEMENT

CELLULAR INFILTRATE

FOURTH PREMOLARS

Specimen #3

Dog #22 - Mandibular Right - Distal Root

Section #	N	B	L	T	I	C
1	+					+
2	+					+
3	+					+
4					+	+
5				+	+	+
6				+	+	+
7			+	+	+	+
8			+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11*		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+	+	+	+	+
17			+	+	+	+
18			+	+	+	+
19			+	+	+	+
20			+	+	+	+
21				+	+	+
22					+	+
23						+
24						+
25						+

*Apical Center

TABLE IX

PERIAPICAL BONE INVOLVEMENT

CELLULAR INFILTRATE

FOURTH PREMOLARS

Specimen #8

Dog #22 - Mand. Left - Distal Root

Sec. #	N	B	L	T	I	C
1						+
2						+
3				+	+	+
4				+	+	+
5		+		+	+	+
6		+		+	+	+
7		+	+	+	+	+
8		+	+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+	+	+	+	+
15 *		+	+	+	+	+
16		+	+	+	+	+
17		+		+	+	+
18		+		+	+	+
19		+		+	+	+
20				+	+	+
21				+	+	+
22					+	+
23						+
24						+
25	+					
26	+					
27	+					

Specimen #10

Dog #14 - Mand. Left - Distal Root

Sec. #	N	B	L	T	I	C
1						+
2					+	+
3					+	+
4				+	+	+
5		+	+	+	+	+
6		+	+	+	+	+
7		+	+	+	+	+
8		+	+	+	+	+
9 *		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+		+	+	+
15		+		+	+	+
16		+		+	+	+
17				+	+	+
18				+	+	+

*Apical Center

TABLE X

PERIAPICAL BONE INVOLVEMENT -

CELLULAR INFILTRATE

FOURTH PREMOLARS

Specimen #17

Dog #14 - Mand. Right - Distal Root

Sec. #	N	B	L	T	I	C
1				+	+	+
2				+	+	+
3			+	+	+	+
4			+	+	+	+
5		+	+	+	+	+
6		+	+	+	+	+
7		+	+	+	+	+
8		+	+	+	+	+
9 *		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14			+	+	+	+
15			+	+	+	+
16			+	+	+	+
17			+	+	+	+
18			+	+	+	+
19				+	+	+
20			+	+	+	+
21				+	+	+
22					+	+
23					+	+
24					+	+
25						+

Specimen #18

Dog #19 - Mand. Right - Distal Root

Sec. #	N	B	L	T	I	C
1					+	+
2				+	+	+
3				+	+	+
4		+	+	+	+	+
5 *		+	+	+	+	+
6		+	+	+	+	+
7		+	+	+	+	+
8		+	+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13			+	+	+	+
14				+	+	+
15					+	+
16					+	+
17						?
18						?

*Apical Center

TABLE XI

PERIAPICAL BONE INVOLVEMENT -

CELLULAR INFILTRATE

FOURTH PREMOLARS

Specimen #4

Dog #22 - Mand. Right - Mesial Root

Sec. #	N	B	L	T	I	C
1					+	+
2					+	+
3					+	+
4				+	+	+
5				+	+	+
6				+	+	+
7		+		+	+	+
8		+		+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13 *		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+	+	+	+	+
17		+	+	+	+	+
18			+	+	+	+
19		+	+	+	+	+
20		+	+	+	+	+
21				+	+	+
22				+	+	+
23				+	+	+
24					+	+
25					+	+
26					+	+

Specimen #9

Dog #22 - Mand. Left - Mesial Root

Sec. #	N	B	L	T	I	C
1					+	+
2					+	+
3		+		+	+	+
4		+		+	+	+
5		+	+	+	+	+
6		+	+	+	+	+
7		+	+	+	+	+
8		+	+	+	+	+
9 *		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+	+	+	+	+
15		+		+	+	+
16		+		+	+	+
17		+		+	+	+
18		+		+	+	+
19				+	+	+
20					+	+
21						+
22						+

*Apical Center

TABLE XII

PERIAPICAL BONE INVOLVEMENT - CELLULAR INFILTRATE FOURTH PREMOLARS

Specimen #16

Dog #14 - Mand. Right - Mesial Root

Sec. #	N	B	L	T	I	C
1					+	+
2				+	+	+
3				+	+	+
4		+		+	+	+
5		+	+	+	+	+
6		+	+	+	+	+
7 *		+	+	+	+	+
8		+	+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11			+	+	+	+
12			+	+	+	+
13				+	+	+
14				+	+	+
15				+	+	+
16					+	+
17					+	+
18					+	+
19					+	+
20						+

Specimen #19

Dog #19 - Mand. Right - Mesial Root

Sec. #	N	B	L	T	I	C
1				+	+	+
2		+		+	+	+
3		+		+	+	+
4		+	+	+	+	+
5		+	+	+	+	+
6		+	+	+	+	+
7		+	+	+	+	+
8		+	+	+	+	+
9 *		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+		+	+	+
14		+		+	+	+
15				+	+	+
16					+	+
17					+	+
18						+
19						+

*Apical Center

TABLE XIII

PERIAPICAL BONE INVOLVEMENT -

CELLULAR INFILTRATE

FOURTH PREMOLAR

Specimen #11

Dog #14 - Mand. Left - Mesial Root

Sec. #	N	B	L	T	I	C
1						POOR FIXATION
2						
3						
4						
5						
6						
7						
8						
9						
10		+?	+?	+	+	+
11		+?	+?	+	+	+
12		+	+	+	+	+
13 *		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+	+	+	+	+
17		+	+	+	+	+
18		+	+	+	+	+
19		+	+	+	+	+
20		+	+	+	+	+
21		+	+	+	+	+
22			+	+	+	+
23					+	+
24					+	+
25						+
26						+
27						+
28						+

FIRST MOLAR

Specimen #20

Dog #19 - Mand. Right - Mesial Root

Sec. #	N	B	L	T	I	C
1						+
2						+
3					+	+
4					+	+
5			+	+	+	+
6			+	+	+	+
7			+	+	+	+
8			+	+	+	+
9			+	+	+	+
10			+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+	+	+	+	+
15 *		+	+	+	+	+
16		+	+	+	+	+
17		+	+	+	+	+
18		+	+	+	+	+
19		+	+	+	+	+
20		+	+	+	+	+
21		+	+	+	+	+
22			+	+	+	+
23			+	+	+	+
24			+	+	+	+
25				+	+	+
26				+	+	+
27				+	+	+

*Apical Center

TABLE XIV

PERIAPICAL BONE INVOLVEMENT - CELLULAR INFILTRATE FIRST MOLARS

Specimen #5

Dog #22 - Mand. Right - Mesial Root

Sec. #	N	B	L	T	I	C
1					+	+
2			+	+	+	+
3				+	+	+
4				+	+	+
5			+	+	+	+
6		+	+	+	+	+
7		+	+	+	+	+
8		+	+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11 *		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+	+	+	+	+
17		+	+	+	+	+
18		+	+	+	+	+
19		+	+	+	+	+
20		+	+	+	+	+
21		+	+	+	+	+
22		+	+	+	+	+
23		+	+	+	+	+
24				+	+	+
25					+	+
26					+	+
27					+	+
28						+
29						+
30						+
31	+					
32	+					

Specimen #7

Dog #22 - Mand. Left - Mesial Root

Sec. #	N	B	L	T	I	C
1		+	+	+	+	+
2		+	+	+	+	+
3		+	+	+	+	+
4		+	+	+	+	+
5		+	+	+	+	+
6		+	+	+	+	+
7 *		+	+	+	+	+
8		+	+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+	+	+	+	+
17		+	+	+	+	+
18		+	+	+	+	+
19		+	+	+	+	+
20				+	+	+
21					+	+
22					+	+
23					+	+
24						+
25						+
26						+
27						?
28						?

*Apical Center

TABLE XV

PERIAPICAL BONE INVOLVEMENT -

CELLULAR INFILTRATE

FIRST MOLARS

Specimen #13

Dog #14 - Mand. Left - Mesial Root

Sec. #	N	B	L	T	I	C
1						
2						
3					+	+
4				+	+	+
5			+	+	+	+
6			+	+	+	+
7			+	+	+	+
8		+	+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+	+	+	+	+
17		+	+	+	+	+
18		+	+	+	+	+
19		+	+	+	+	+
20		+	+	+	+	+
21			+	+	+	+
22			+	+	+	+
23				+	+	+
24				+	+	+
25				+	+	+
26					+	+
27					+	+
28					+	+
29					+	+
30						?

Specimen #14

Dog #14 - Mand. Right - Mesial Root

Sec. #	N	B	L	T	I	C
1					+	+
2				+	+	+
3			+	+	+	+
4		+	+	+	+	+
5		+	+	+	+	+
6		+	+	+	+	+
7		+	+	+	+	+
8		+	+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13 *		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+	+	+	+	+
17		+	+	+	+	+
18		+	+	+	+	+
19		+	+	+	+	+
20			+	+	+	+
21			+	+	+	+
22				+	+	+
23				+	+	+
24				+	+	+
25				+	+	+
26					+	+
27					+	+
28						+
29						+
30						+

*Apical Center

TABLE XVI

PERIAPICAL BONE INVOLVEMENT - CELLULAR INFILTRATE FIRST MOLARS

Specimen #6

Dog #22 - Mand. Left - Distal Root

Sec. #	N	B	L	T	I	C
1						+
2					+	+
3				+	+	+
4				+	+	+
5			+	+	+	+
6			+	+	+	+
7		+	+	+	+	+
8		+	+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13 *		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+	+	+	+	+
17		+	+	+	+	+
18		+	+	+	+	+
19		+	+	+	+	+
20		+	+	+	+	+
21		+	+	+	+	+
22		+	+	+	+	+
23		+	+	+	+	+
24			+	+	+	+
25				+	+	+
26				+	+	+
27					+	+
28						+
29						+

*Apical Center

Specimen #12

Dog #14 - Mand. Left - Distal Root

Sec. #	N	B	L	T	I	C
1						+
2						+
3						+
4				+	+	+
5			+	+	+	+
6			+	+	+	+
7			+	+	+	+
8			+	+	+	+
9			+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+	+	+	+	+
17		+	+	+	+	+
18		+	+	+	+	+
19 *		+	+	+	+	+
20		+	+	+	+	+
21		+	+	+	+	+
22		+	+	+	+	+
23		+	+	+	+	+
24		+	+	+	+	+
25		+	+	+	+	+
26		+	+	+	+	+
27		+	+	+	+	+
28		+	+	+	+	+
29		+	+	+	+	+
30			+	+	+	+
31			+	+	+	+
32				+	+	+
33				+	+	+
34				+	+	+
35					?	+
36					?	+
37					?	+
38					?	+

TABLE XVII

PERIAPICAL BONE INVOLVEMENT - CELLULAR INFILTRATE FIRST MOLARS

Specimen #15

Dog #14 - Mand. Right - Distal Root

Sec. #	N	B	L	T	I	C
1					?	+
2					+	+
3					+	+
4					+	+
5					+	+
6				+	+	+
7 *				+	+	+
8		+	+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+	+	+	+	+
17		+	+	+	+	+
18			+	+	+	+
19			+	+	+	+
20				+	+	+
21				+	+	+
22				+	+	+
23				+	+	+
24				+	+	+
25				+	+	+
26					+	+
27					+	+

Specimen #21

Dog #19 - Mand. Right - Distal Root

Sec. #	N	B	L	T	I	C
1						
2						
3						
4				+	+	+
5		+		+	+	+
6		+	+	+	+	+
7 *		+	+	+	+	+
8		+	+	+	+	+
9		+	+	+	+	+
10		+	+	+	+	+
11		+	+	+	+	+
12		+	+	+	+	+
13		+	+	+	+	+
14		+	+	+	+	+
15		+	+	+	+	+
16		+		+	+	+
17		+		+	+	+
18		+		+	+	+
19				+	+	+
20				+	+	+
21				+	+	+
22					+	+
23					+	+
24						+
25						+

*Apical Center

APPROVAL SHEET

The thesis submitted by Lance W. Crawford, D.D.S., has been read and approved by the following committee:

Dr. Franklin S. Weine, Director
Professor, Endodontics, Loyola University

Dr. Norman K. Wood
Professor, Oral Diagnosis, Loyola University

Dr. Thomas E. Emmering
Associate Professor, Radiology, Loyola University

The final copies have been examined by the director of the thesis and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the thesis is now given final approval by the committee with reference to content and form.

The thesis is therefore accepted in partial fulfillment of the requirements for the degree of Master of Science in Oral Biology.

6-9-87
Date

Franklin S. Weine
Director's Signature